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RESPIRATORY CARE

A MONTHLY SCIENCE JOURNAL
37TH YEAR—ESTABLISHED 1956

- Factors Affecting Lung Volume Changes: A Bench Study
- Exhaled CO_2 To Assess ET-Tube Placement
- Effect on Subglottic Pressure of Upper-Airway Bypass
- Smooth-Muscle Regulation: The Role of Inhaled Nitric Oxide Gas
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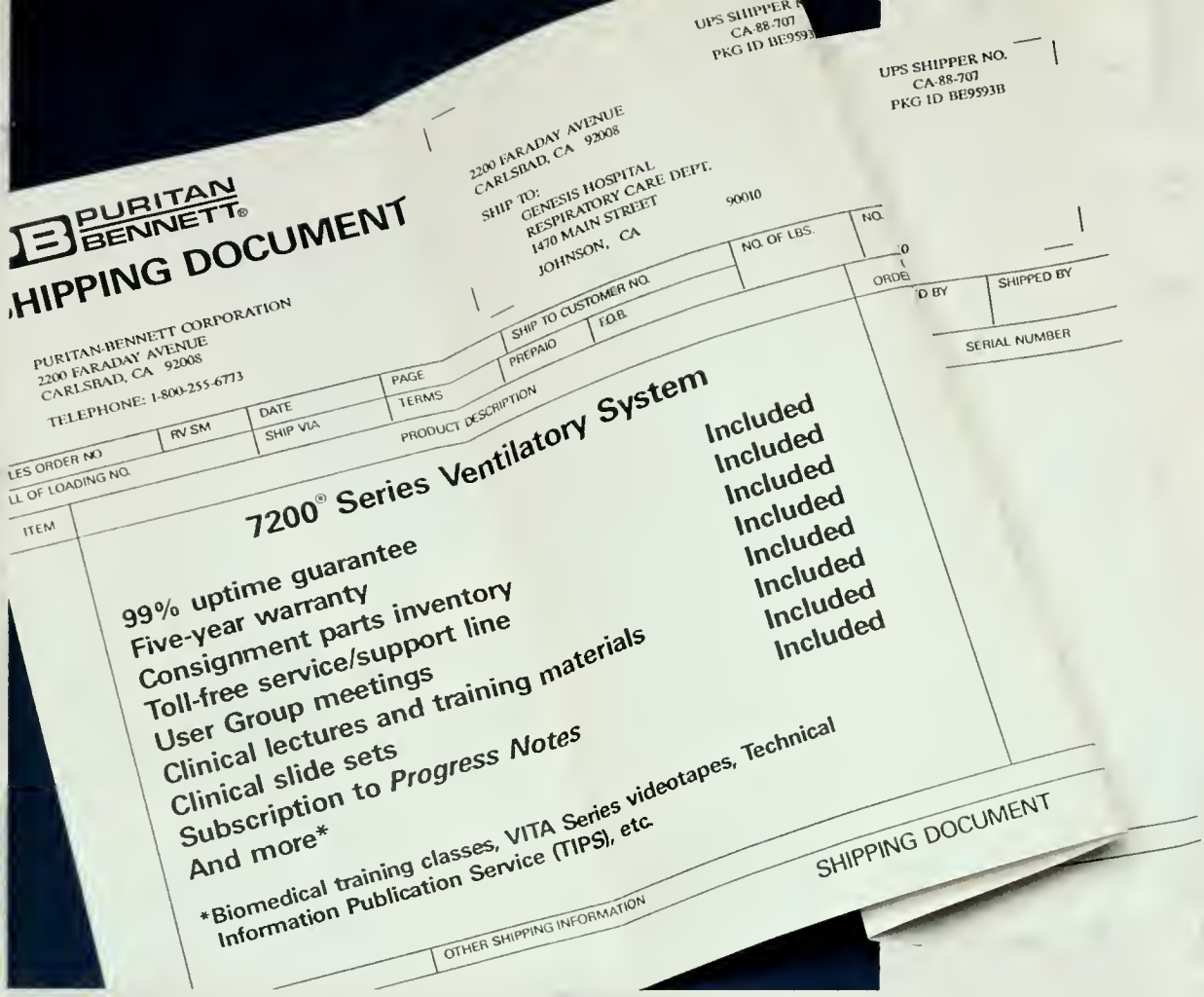

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CONTENTS

October 1992
Volume 37, Number 10

ORIGINAL CONTRIBUTIONS

1153 Factors Affecting Lung Volume Changes during Newborn Mechanical Ventilation: A Bench Study
by Robert M Lewis—Chicago, Illinois

1161 Rapid Analysis of Exhaled CO_2 To Assess Endotracheal Tube Placement
by Sharon Day, Larry Wooton, and Neil R MacIntyre—Durham, North Carolina

1166 The Effect of Partial Upper-Airway Bypass on Subglottic Pressures during Acute Lung Injury in Sheep
by Marin H Kollef, Michael T McCormack, and Ron E Banks—St Louis, Missouri, and Aurora, Colorado

REVIEWS, OVERVIEWS, & UPDATES

1175 Pulmonary Vascular Smooth-Muscle Regulation: The Role of Inhaled Nitric Oxide Gas
by Christopher C Miller and John WR Miller—Vancouver, British Columbia, Canada, and Santa Ana, California

CONTINUING EDUCATION EXAMINATION

1212 ***CRCE through the Journal:*** Answer Key

PFT CORNER

1187 PFT Corner #45—Asthma and Vocal Cord Dysfunction Syndrome: When Wheezing Is Hysterical
by Jack Wanger and William R Beam—Denver, Colorado

TEST YOUR RADIOLOGIC SKILL

1193 Is This a Simple Case of Respiratory Distress Syndrome?
by Katherine A Douglas and Kathleen S Carlson—Toledo, Ohio

MURDER MYSTERY

1197 "Murder Mystery" for Student Practice of Pulmonary Physiology Calculations
by Michael B Maron and Frank J Bosso—Rootstown, Ohio

BOOKS, FILMS, TAPES, & SOFTWARE REVIEWS

1204 Winning in Your Profession by Writing Books—A How-To Book for Professionals in the Biomedical and Behavioral Sciences and the Law, by Bowen Hosford
reviewed by Thomas A Barnes—Boston, Massachusetts

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CONTENTS, *Continued*

October 1992
Volume 37, Number 10

1205 Atlas of Bronchoscopy: Technique, Diagnosis, Differential Diagnosis, and Therapy, by Heinrich D Becker MD, et al (translated by Thomas J DeKornfeld MD and Heinrich D Becker MD)
reviewed by John W Shigeoka—Salt Lake City, Utah

1205 Hemodynamics and Gas Exchange: A Clinical Guide, by Paul Ouellet RRT
reviewed by James A Ganetis—Stony Brook, New York

1206 Pediatric Home Care: Results of a National Evaluation of Programs for Ventilator-Assisted Children, by LuAnn Aday PhD, Marlene J Aitken MAMS OTR/L, and Donna Hope Wegener MA
reviewed by Darlene D Cimo—Metairie, Louisiana

1207 Respiratory Infections in the Elderly, edited by Michael S Niederman
reviewed by Bruce P Krieger—Miami Beach, Florida

LETTERS

1209 Objections to Postural Drainage Guideline
by Colleen M Kigin—Boston, Massachusetts; with response by Dean Hess and Pat Brougher—York, Pennsylvania, and Dallas, Texas

1211 Bug? Yes. Insect? No.
by David J Pierson—Seattle, Washington; with response by Pat Brougher—Dallas, Texas

1211 Baggers, Not Beggars
by Tom Burns—Phoenix, Arizona

CORRECTIONS

1195 Corrections to Clinical Practice Guidelines: Exercise Testing for Evaluation of Hypoxemia and/or Desaturation (Respir Care 1992;37:907-912) and Oxygen Therapy in the Home or Extended Care Facility (Respir Care 1992;37:918-922)

1195 Corrections to Hess D and Eitel D. Monitoring during Resuscitation (Respir Care 1992;37:739-768)

ABSTRACTS

1142 Summaries of Pertinent Articles from Other Journals

CALENDAR OF EVENTS

1214 Meeting Dates, Locations, Themes

NOTICES

1213 Examination Dates, Notices, Prizes

INDEXES

1216 Authors in This Issue

1216 Advertisers in This Issue

1147 Advertiser Help Lines

Editorials , Statements, and Reviews To Note

Fluid Management Strategy in Acute Lung Injury (editorial)—LD Hudson. Am Rev Respir Dis 1992;145:988.

Drug-Induced Pulmonary Disease: An Update (review)—EC Rosenow III, JL Myers, SJ Swensen, RJ Pisani. Chest 1992;102:239.

Cigarette Advertising and Corporate Responsibility (editorial)—S Broder. JAMA 1992;268(6):782.

Airway Hyperresponsiveness and Airway Wall Thickening in Asthma: A Quantitative Approach (editorial)—RD Kamm, JM Drazen. Am Rev Respir Dis 1992;145:1249.

Environmental Lung Diseases: Relationship between Acute Inflammatory Responses to Air Pollutants and Chronic Lung Disease (NHLBI workshop summary)—J Crapo, FJ Miller, B Mossman, WA Pryor, JP Kiley. Am Rev Respir Dis 1992;145:1506.

Inhaled Therapy in Infants: Why Not Nebulize Glucocorticoids? (editorial)—AK Kamada, SJ Szefler. Pediatr Pulmonol 1992;13:198.

A Prospective Study of Cigarette Smoking and Risk of Cataract in Men—WG Christen, JE Manson, JM Seddon, RJ Glynn, JE Buring, B Rosner, CH Hennekens. JAMA 1992; 268:989.

OBJECTIVE: To examine the association between cigarette smoking and the incidence of cataract. DESIGN, SETTING, & PARTICIPANTS: The design was a prospective cohort study using data from the Physicians' Health Study, a randomized trial of aspirin and beta carotene among 22,071 U.S. male physicians aged 40 to 84 years that began in 1982. This analysis includes the 17,824 physicians who did not report cataract at baseline and did provide complete risk factor information. Based on information reported at baseline, 10% were current smokers, 39% were past smokers, and 51% were never smokers. MAIN OUTCOME MEASURE: An incident cataract was defined as a self-report confirmed by medical record review to have been first diagnosed after randomization, age-related in origin, and responsible for a decrease in best corrected visual acuity to 20/30 or worse. MAIN RESULTS: During 60 months of follow-up, 557 incident cataracts among 371 participants were confirmed. Compared with never smokers, current smokers of 20 or more cigarettes per day had a statistically significant increase in the risk of cataract (relative risk [RR], 2.16; 95% confidence interval [CI], 1.46-3.20; $p < 0.001$). Similar results were obtained after simultaneously controlling for other potential cataract risk factors in a logistic regression model (RR, 2.05; 95% CI, 1.38-3.05; $p < 0.001$). Among the 557 eyes with cataract, nuclear sclerotic changes were present in 442 while posterior subcapsular changes were present in 204. After controlling for other potential cataract risk factors, current smokers of 20 or more cigarettes per day had statistically significant increases in nuclear sclerosis (RR, 2.24; 95% CI, 1.47-3.41; $p < 0.001$) and posterior subcapsular (RR, 3.17; 95% CI, 1.81-5.53; $p < 0.001$) cataract. Past smokers had an elevated risk of posterior subcapsular (RR, 1.44; 95% CI, 0.97-2.13; $p = 0.07$) but not nuclear sclerosis cataract. For current smokers of

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DURING THE DOSING PROCEDURE, TRANSIENT EPISODES OF BRADYCARDIA AND DECREASED OXYGEN SATURATION HAVE BEEN REPORTED. If these occur, stop the dosing procedure and initiate appropriate measures to alleviate the condition. After stabilization, resume the dosing procedure.

PRECAUTIONS

General

Rales and moist breath sounds can occur transiently after administration. Endotracheal suctioning or other remedial action is not necessary unless clear-cut signs of airway obstruction are present.

Increased probability of post-treatment nosocomial sepsis in SURVANTA-treated infants was observed in the controlled clinical trials (Table 3). The increased risk for sepsis among SURVANTA-treated infants was not associated with increased mortality among these infants. The causative organisms were similar in treated and control infants. There was no significant difference between groups in the rate of post-treatment infections other than sepsis.

Use of SURVANTA in infants less than 600 g birth weight or greater than 1750 g birth weight has not been evaluated in controlled trials. There is no controlled experience with use of SURVANTA in conjunction with experimental therapies for RDS (eg, high-frequency ventilation or extracorporeal membrane oxygenation).

No information is available on the effects of doses other than 100 mg phospholipids/kg, more than four doses, dosing more frequently than every 6 hours, or administration after 48 hours of age.

Carcinogenesis, Mutagenesis, Impairment of Fertility

Reproduction studies in animals have not been completed. Mutagenicity studies were negative. Carcinogenicity studies have not been performed with SURVANTA.

ADVERSE REACTIONS

The most commonly reported adverse experiences were associated with the dosing procedure. In the multiple-dose controlled clinical trials, transient bradycardia occurred with 11.9% of doses. Oxygen desaturation occurred with 9.8% of doses.

Other reactions during the dosing procedure occurred with fewer than 1% of doses and included endotracheal tube reflux, pallor, vasoconstriction, hypotension, endotracheal tube blockage, hypertension, hypocarbia, hypercarbia, and apnea. No deaths occurred during the dosing procedure, and all reactions resolved with symptomatic treatment.

The occurrence of concurrent illnesses common in premature infants was evaluated in the controlled trials. The rates in all controlled studies are in Table 3.

TABLE 3

	All Controlled Studies		
Concurrent Event	SURVANTA (%)	Control (%)	P-Value[a]
Patent ductus arteriosus	46.9	47.1	0.814
Intracranial hemorrhage	48.1	45.2	0.241
Severe intracranial hemorrhage	24.1	23.3	0.693
Pulmonary air leaks	10.9	24.7	<0.001
Pulmonary interstitial emphysema	20.2	38.4	<0.001
Necrotizing enterocolitis	6.1	5.3	0.427
Apnea	65.4	59.6	0.283
Severe apnea	46.1	42.5	0.114
Post-treatment sepsis	20.7	16.1	0.019
Post-treatment infection	10.2	9.1	0.345
Pulmonary hemorrhage	7.2	5.3	0.166

[a]P-value comparing groups in controlled studies

When all controlled studies were pooled, there was no difference in intracranial hemorrhage. However, in one of the single-dose rescue studies and one of the multiple-dose prevention studies, the rate of intracranial hemorrhage was significantly higher in SURVANTA patients than control patients (63.3% *v* 30.8%, $P = 0.001$; and 48.8% *v* 34.2%, $P = 0.047$, respectively). The rate in a Treatment IND involving approximately 4400 infants was lower than in the controlled trials.

In the controlled clinical trials, there was no effect of SURVANTA on results of common laboratory tests: white blood cell count and serum sodium, potassium, bilirubin, creatinine.

More than 3700 pretreatment and post-treatment serum samples were tested by Western Blot immunoassay for antibodies to surfactant-associated proteins SP-B and SP-C. No IgG or IgM antibodies were detected.

Several other complications are known to occur in premature infants. The following conditions were reported in the controlled clinical studies. The rates of the complications were not different in treated and control infants, and none of the complications were attributed to SURVANTA.

Respiratory: lung consolidation, blood from the endotracheal tube, deterioration after weaning, respiratory decompensation, subglottic stenosis, paralyzed diaphragm, respiratory failure.

Cardiovascular: hypotension, hypertension, tachycardia, ventricular tachycardia, aortic thrombosis, cardiac failure, cardiorespiratory arrest, increased apical pulse, persistent fetal circulation, air embolism, total anomalous pulmonary venous return.

Gastrointestinal: abdominal distention, hemorrhage, intestinal perforations, volvulus, bowel infarct, feeding intolerance, hepatic failure, stress ulcer.

Renal: renal failure, hematuria.

Hematologic: coagulopathy, thrombocytopenia, disseminated intravascular coagulation.

Central Nervous System: seizures.

Endocrine/Metabolic: adrenal hemorrhage, inappropriate ADH secretion, hyperphosphatemia.

Musculoskeletal: inguinal hernia.

Systemic: fever, deterioration.

Follow-Up Evaluations

To date, no long-term complications or sequelae of SURVANTA therapy have been found.

Single-Dose Studies

Six-month adjusted-age follow-up evaluations of 232 infants (115 treated) demonstrated no clinically important differences between treatment groups in pulmonary and neurologic sequelae, incidence or severity of retinopathy of prematurity, rehospitalizations, growth, or allergic manifestations.

Multiple-Dose Studies

Six-month adjusted age follow-up evaluations have not been completed. Preliminarily, in 605 (333 treated) of 916 surviving infants, there are trends for decreased cerebral palsy and need for supplemental oxygen in SURVANTA infants. Wheezing at the time of examination tended to be more frequent among SURVANTA infants, although there was no difference in bronchodilator therapy.

Twelve-month follow-up data from the multiple-dose studies have been completed in 328 (171 treated) of 909 surviving infants. To date no significant differences between treatments have been found, although there is a trend toward less wheezing in SURVANTA infants in contrast to the six month results.

OVERDOSAGE

Overdosage with SURVANTA has not been reported. Based on animal data, overdosage might result in acute airway obstruction. Treatment should be symptomatic and supportive.

Rales and moist breath sounds can transiently occur after SURVANTA is given, and do not indicate overdosage. Endotracheal suctioning or other remedial action is not required unless clear-cut signs of airway obstruction are present.

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fewer than 20 cigarettes per day, no increased risks were observed of total, nuclear sclerosis, or posterior subcapsular cataract. CONCLUSIONS: These data provide support for the hypothesis that cigarette smoking increases the risk of developing both nuclear sclerosis and posterior subcapsular cataract.

A Prospective Study of Cigarette Smoking and Risk of Cataract Surgery in Women—SE Hankinson, WC Willett, GA Colditz, JM Seddon, B Rosner, FE Speizer, MJ Stampfer. JAMA 1992;268:994.

OBJECTIVE: To examine prospectively the association between cigarette smoking and the risk of cataract extraction in women. DESIGN: Prospective cohort study beginning in 1980 with 8 years of follow-up. SETTING: United States, multistate. PARTICIPANTS: Registered nurses 45 to 67 years of age and free of diagnosed cancer. There were 50,828 women included in 1980 and others were added as they became 45 years of age. MAIN OUTCOME MEASURE: Incidence of senile cataract extraction. RESULTS: We documented 493 incident senile cataracts diagnosed and extracted during 470,302 person-years of follow-up. The age-adjusted relative risk (RR) among women who smoked at least 65 pack-years was 1.63 (95% confidence interval [CI], 1.18-2.26; p for trend, 0.02). A similar increase in RR was noted when smoking was assessed in terms of number of cigarettes currently smoked or number of cigarettes smoked by past smokers. Results were also similar after adjusting for other potential risk factors for cataract. Smoking was also strongly associated with posterior subcapsular cataract specifically (RR, 2.59; 95% CI, 1.49-4.50 for ≥ 65-pack-year smokers relative to nonsmokers). CONCLUSION: Smoking appears to increase the risk of cataract extraction in women.

Evaluation of Risk Factors for Laryngeal Edema after Tracheal Extubation in Adults and Its Prevention by Dexamethasone: A Placebo-Controlled, Double-Blind, Multicenter Study—J-Y Darmon, A Rauss, D Dreyfuss, G Bleichner, D Elkharrat, B Schlemmer, et al. Anesthesiology 1992;77:245.

Because laryngeal edema (LE) after tracheal extubation is likely to result from an exudative response, corticosteroids often are given routinely as a preventive treatment. No adequate controlled study supports this strategy, however. A prospective, randomized, placebo-controlled, double-blind, multicenter trial that included 700 consecutive patients requiring tracheal intubation and mechanical ventilation was conducted to determine risk factors for LE occurrence after tracheal extubation in adults and to evaluate the efficacy of corticosteroids in its prevention. One hour before extubation, patients were given either an intravenous bolus of 8 mg dexamethasone or a placebo. Patients were divided into two groups: (1) those in whom short-duration intubation (SDI, less than 36 h) was administered; and (2) those in whom long-duration intubation (LDI, more than 36 h) was administered. Minor LE was diagnosed when either stridor or laryngeal dyspnea, or both, occurred; major LE was diagnosed when reintubation due to LE was required, with LE evidenced during direct laryngoscopy. The overall incidence of LE was 4.2% and varied among the six participating centers from 2.3 to 6.9% (not significant). In only seven patients (1%), all with LDI, was tracheal reintubation required for LE. Laryngeal edema occurred more frequently after LDI than after SDI (7.2 vs 0.9%; $p < 0.001$). It also was more frequent in female than in male patients (20/284 vs 8/379; $p < 0.05$), irrespective of intubation duration and treatment. There was no association between LE and either difficulty/route of intubation or admission diagnosis. Its overall incidence was not different between patients given dexamethasone and those given placebo, whether they were in the SDI or LDI group (0.7 vs 1.2% and 5.7 vs 8.7%, respectively). Dexamethasone did not affect the number of patients reintubated. Assuming an incidence of LE in each group similar to that found in our study, more than 7,000 patients would need to be studied to evaluate adequately the presence of a Type-2 error. Therefore, we conclude that the overall incidence of postextubation LE in adults is small and that risk factors for LE include an intubation duration of more than 36 h and female gender. In addition, 8 mg dexamethasone given as an intravenous bolus 1 h before extubation does not appear to prevent either minor or major forms of LE.

Protection from Environmental Tobacco Smoke in California: The Case for a Smoke-Free Workplace—R Borland, JP Pierce, DM Burns, E Gilpin, M Johnson, D Bal. JAMA 1992;268:749.

OBJECTIVE: To determine the extent of exposure of nonsmoking indoor workers to environmental tobacco smoke (ETS) according to type of work-site smoking policy, work area, workplace size, and demographic characteristics. DESIGN & PARTICIPANTS: Participants included 7,162 adult, nonsmoking, indoor workers who were interviewed as part of the 1990 California Tobacco Survey. Respondents were asked whether anyone had smoked in their work area within the past 2 weeks. RESULTS: An estimated 2.2 million California nonsmokers were exposed to tobacco smoke at indoor work sites in 1990. Nonsmoker exposure to ETS was 9.3% for those

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working in a smoke-free worksite, 23.2% for those working where there was only a work-area restriction, 46.7% for those working where there was a policy that did not include the work area, and 51.4% for those working where there was no worksite smoking policy. After adjustment for type of work area (eg, office, open area), workplace size, and demographic factors, it was determined that nonsmokers working where there was only a work-area ban were 2.8 times more likely to be exposed to ETS than those working in a smoke-free work site. In workplaces with no policy or a policy not covering the work area, nonsmokers were over eight times more likely to be exposed to ETS than those who worked in a smoke-free work site. Nonsmokers who were 18 to 24 years of age, male, or Hispanic, and those with less than a high school education had more exposure to ETS. CONCLUSION: These results indicate that adequate protection of nonsmokers from ETS exposure requires a smoke-free work site.

Impact of Dyspnea and Physiologic Function on General Health Status in Patients with Chronic Obstructive Pulmonary Disease—DA Mahler, K Faryniarz, D Tomlinson, GL Colice, AG Robins, EM Olmstead, GT O'Connor. Chest 1992; 102:395.

STUDY OBJECTIVE: To examine the relationships among clinical dyspnea ratings, physiologic pulmonary function, and general health status in symptomatic patients with chronic obstructive pulmonary disease (COPD). DESIGN: Observational data collected at a baseline state. SETTING: Outpatient pulmonary disease clinics at a university hospital and two VA medical centers. PATIENTS: 110 male patients with COPD with no significant comorbidity were recruited. MEASUREMENTS & RESULTS: Clinical ratings of dyspnea were measured by the multidimensional baseline dyspnea index (BDI). Pulmonary function tests included forced vital capacity (FVC), forced expiratory volume in 1 s (FEV_1), and maximal inspiratory mouth pressure (P_{Imax}). General health status was assessed with the Medical Outcomes Study short-form survey, which consists of 20 questions that cover 6 health components. The mean age of the patients was 67 ± 8 yr (± SD). The mean value for FVC was 2.84 ± 0.84 L (68 ± 18% of predicted), for FEV_1 was 1.28 ± 0.59

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L (44 ± 17% of predicted), and for P_{Imax} was 59.0 ± 25.0 cm H_2O. The BDI score and P_{Imax} were significantly correlated with 5 of the 6 components of general health status. Only 3 of the 6 components of general health were significantly correlated with FEV_1 as % predicted (r_s value range, 0.30-0.44) and with FVC as % predicted (r_s value range, 0.25-0.33). Statistical comparisons showed that the BDI score had significantly higher correlations than FVC (% predicted), FEV_1 (% predicted), and P_{Imax} values with physical functioning and role functioning. Multiple linear regression analysis showed that the BDI score was the only statistically significant predictor of role functioning, mental health, and health perceptions for general health status, whereas both the BDI score and FEV_1 (% predicted) were independent predictors of physical functioning and social functioning. CONCLUSION: Dyspnea ratings influence and predict general health status to a greater extent than do physiologic measurements in symptomatic patients with COPD. A shift in focus from the pathophysiology of disease to assessment and relief of symptoms may provide more meaningful benefits for the individual patient in terms of quality of life. This consideration requires that healthcare providers use available measuring tools in clinical practice to quantify symptoms, as well as overall health status.

The Diagnosis of Potentially Fatal Asthma in Hospitalized Adults: Patient Characteristics and Increased Severity of Asthma—TP Miller, PA Greenberger, R Patterson. Chest 1992;102:515.

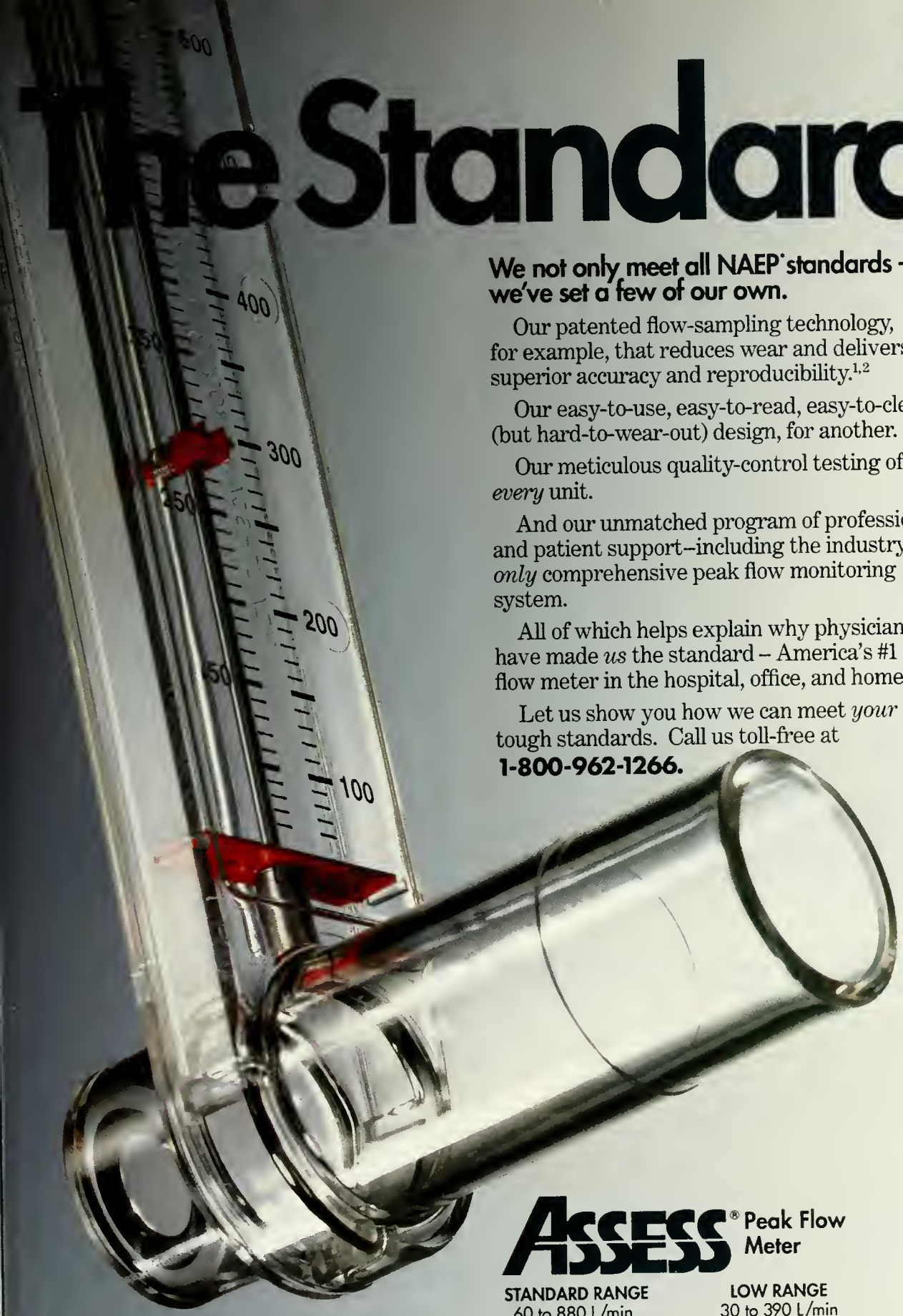
We evaluated various patient characteristics in patients hospitalized for asthma during 1987 to 1990. Potentially fatal asthma (PFA) was identified in 26 of 87 adult patients (29.9%) hospitalized. Patients with PFA had increased frequency of prednisone use prior to hospitalization ($p < 0.001$), shorter duration of symptoms before hospitalization ($p < 0.001$), longer hospitalization ($p < 0.001$), were more likely to have had three or more prior hospitalizations ($p < 0.001$), and had reduced presenting peak expiratory flowrates ($p < 0.05$). Major psychiatric diagnoses and noncompliance were significantly related to PFA cases. The diagnosis of PFA identifies a higher risk patient with asthma. The data suggest that at the time of hospitalization the PFA patient has had a shorter recognized prodrome of increased respiratory symptoms, reduced peak expiratory flowrates, and greater likelihood of major psychiatric disease or noncompliance. Effective ambulatory control of PFA and non-PFA is advisable with earlier use and higher dosages of oral corticosteroids.

Increased Exercise Capacity in Hypoxemic Patients after Long-Term Oxygen Therapy—DA Morrison, JR Stovall. Chest 1992;102:542.

Low-flow continuous oxygen can lead to significant improvement in exercise capacity in selected patients with stable hypoxemic pulmonary disease. Although the mechanisms of improvement are incompletely understood, two tenable hypotheses are (1) the relief of hypoxic pulmonary vasoconstriction and (2) improved peripheral oxygen delivery. This prospective study was performed to examine these two hypotheses. Stable patients with hypoxemic lung disease performed symptom-limited ergometry with hemodynamic monitoring before and after > 1 month of long-term home oxygen therapy. There were 22 patients who increased exercise capacity by > 100 KPM/min and > 4 min duration and 11 patients who did not increase their exercise capacity > 2 min or in KPM/min. Neither the responders nor the nonresponders had significant changes in pulmonary pressures or resistances or effective arterial volume elastances at exercise after long-term O_2 therapy. The responders had significant increases in oxygen delivery during exercise (148 ± 33 mL/min vs 184 ± 51 mL/min, $p = 0.004$), cardiac output (7.7 ± 1.6 L/min vs 8.7 ± 2.1 L/min, $p = 0.003$), and arterial oxygen content (18.6 ± 2.6 mL/dL vs 20.2 ± 2.8 mL/dL, $p = 0.02$). The nonresponders had no significant changes in these parameters during exercise. Increased exercise capacity in response to long-term O_2 therapy is associated with increased O_2 delivery not relief of hypoxic vasoconstriction (in terms of pressure or resistance or arterial elastance). Increased O_2 delivery can accrue from both increased cardiac output and increased arterial O_2 content. Increase in arterial O_2 content is unique to O_2 relative to all vasodilator drugs.

Pulmonary Barotrauma in Mechanical Ventilation: Patterns and Risk Factors—RB Gammon, MS Shin, SE Buchalter. Chest 1992;102:568.

The incidence of mediastinal emphysema (ME) and pneumothorax (PTX) was analyzed to determine the roentgenographic patterns and risk factors for the development of barotrauma in a population of mechanically ventilated patients. The roentgenograms of 139 intubated patients admitted to our medical intensive care unit over a 10-month period were evaluated for the presence of ME and PTX. Barotrauma was diagnosed in 34 of these patients, and ME was the initial manifestation in



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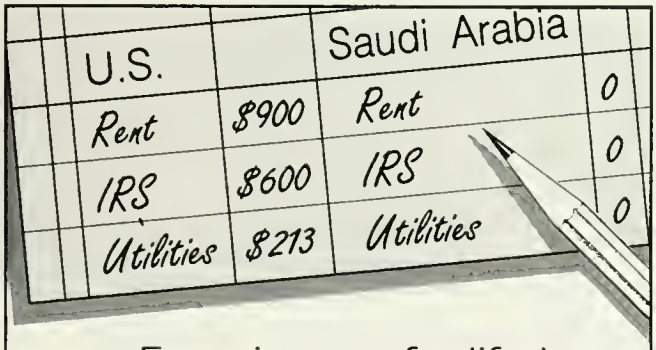
24 patients. Of these patients with initial ME, 10 subsequently developed PTX, a positive predictive value of 42%. The adult respiratory distress syndrome (ARDS) patient population was at highest risk for barotrauma, with an intermediate risk seen in those admitted with COPD or pneumonia. Values of peak inspiratory pressure, positive end-expiratory pressure level, respiratory rate, tidal volume, and minute ventilation were significantly elevated in patients who developed barotrauma as compared with patients who did not develop barotrauma. However, these elevations in part reflect the high incidence of barotrauma in the ARDS population, a patient group in which all of the above parameters were elevated.

Comparison of Helium Dilution and Nitrogen Washout Measurements of Functional Residual Capacity in Infants and Very Young Children—RS Tepper, S Asdell. Pediatr Pulmonol 1992;13:250.

In infants and very young children, functional residual capacity (FRC) is the lung volume most frequently measured, and gas dilution techniques are most frequently used to measure FRC. We compared measurements of FRC by helium dilution (FRC_{He}) and nitrogen washout (FRC_{N_2}) in a lung model of known volumes (20, 40, 60, 80 mL), in 8 normal infants (NL), 8 infants with acute respiratory illness (ARI), and 7 infants with chronic lung disease (CLD). In the model lung, measurements of FRC_{He} had a significantly greater coefficient of variation compared to FRC_{N_2} (6.5 vs 1.5%, $p < 0.02$), but there was no such difference in the results from all infants combined (6.5 vs 6.2%). In the model lung, the difference between known volumes and the measured values was significantly different from zero only for FRC_{He} at 20 mL (–4.4 mL, $p < 0.02$). For both FRC_{He} and FRC_{N_2}, the slopes of the regression equations were 0.99 and the intercepts not significantly different from zero. We conclude that techniques for measuring FRC_{He} and FRC_{N_2} yield accurate, reproducible, and comparable results in normal, healthy infants and very young children, and in those with respiratory disease.



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Factors Affecting Lung Volume Changes during Newborn Mechanical Ventilation: A Bench Study

Robert M Lewis BA RRT

BACKGROUND: The functional residual capacity and tidal volume of mechanically ventilated infants recovering from respiratory distress syndrome may increase as lung compliance improves, perhaps with deleterious effects. I conducted a bench study using 5 infant ventilators in current use to determine factors affecting those volume increases. MATERIALS & METHODS: A lung simulator set at 1.0 mL/cm H_2O [0.01 L/kPa] was attached to each of the 5 infant ventilators tested. Ventilator flow was set at 20 L/min, inspiratory time at 0.66 seconds, and peak inspiratory pressure at 30 cm H_2O. Simulator lung compliance was then increased to 3 mL/cm H_2O [0.03 L/kPa], without any change in ventilator settings. Changes in baseline end-expiratory volume and tidal volume were recorded, and the sum of these two volume changes (called maximum lung volume, or MaxV) was determined. This procedure was repeated, but with the ventilator flow set at the minimum needed to produce PIP of 30 cm H_2O. Then, both procedures were repeated with inspiratory time at 0.17 s. Additional trials were conducted on ventilators offering differing pressure waveform or flow modes. Finally, these experiments were repeated with two ventilators, but with the ventilator's circuit compliance increased. RESULTS: All ventilators produced substantial increases in MaxV when simulator lung compliance increased. The magnitude of change varied widely with ventilator brand and settings (range 210% to 13%, or 4 to 63 mL). Reduction in ventilator flow substantially reduced the degree of volume increase. When inspiratory time was reduced, lung volume increase was limited for most ventilators, but not always as much as when flow was reduced. Increased ventilator-circuit compliance also contributed to volume increase. Tidal volume delivered to the lung simulator prior to compliance increase was affected by reductions in inspiratory time or flow in some but not all ventilators. CONCLUSION: The lung volume change produced as a consequence of an increase in lung compliance varied significantly among the ventilators tested. (Respir Care 1992;37:1153-1160.)

Introduction

Increased functional residual capacity (FRC) secondary to spontaneous improvements in compliance and to auto-PEEP is thought to be common in neonates during recovery from respiratory distress syndrome.[1,2] Patient-related factors, such as increasing time constants (secondary to increased total lung compliance), can contribute to increased FRC.[1,2] Increases in total compliance (lungs + chest wall) can occur following administration of surfactant,[3] treatment with bronchodilators and diuretics,[4] and changes in position.[5] In addition to FRC changes, tidal volume may increase to a potentially clinically important degree as lung compliance improves if ventilator settings remain unchanged.

Factors that can potentially influence the ventilator and lung's response to increased compliance include ventilator design, inspiratory time,[6] and ventilator-circuit compliance.[7] The effect of flow on volume changes secondary to increasing compliance has not been directly demonstrated. There-

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A version of this paper was presented by Mr Lewis at the RESPIRATORY CARE OPEN FORUM during the 1990 AARC Annual Meeting held in New Orleans, Louisiana.

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fore, I attempted to determine whether (1) the ventilator flowrate influences volume changes in the same manner in different ventilator brands; (2) the effects of differing levels of flow are modified by differing inspiratory times; and (3) circuit compliance further modifies the response of ventilators to changing lung mechanics.

Materials and Methods

Lung Simulator

I used a commercially available lung simulator (Bio-Tek VT-2),* which can simulate both an infant with respiratory distress syndrome, or RDS, (lung compliance 1.0 mL/cm H_2O [0.01 L/kPa]) and a recovering infant (compliance 3.0 mL/cm H_2O [0.03 L/kPa]). Airway and lung pressures, delivered tidal volume, and end-expiratory volume produced by PEEP or auto-PEEP (if present) can be measured with this device. (End-expiratory volume as I use the term is defined as the change in the simulator's functional residual capacity, or end-expiratory volume, caused by PEEP or auto-PEEP.) The maximum lung volume, or MaxV, then is the change in end-expiratory volume plus tidal volume and can be calculated. Maximum lung volume includes the contributions of both PEEP and tidal volume and represents the gas contained in the lung as a result of the ventilator. A parabolic resistor, which was calibrated to produce a resistance of 50 cm $H_2O \cdot s \cdot L^{-1}$ [4.9 kPa $\cdot$ s $\cdot$ L^{-1}] at a flow of 40 L/min, was used at both compliance settings.

Ventilators

The ventilators studied were the Siemens Servo 900C, Bourns BP-200, Bear Cub, Sechrist IV 100B, and the InfantStar Ventilator. An identical low-compliance ventilator circuit (0.2 mL/cm H_2O [0.02 L/kPa]) was used for each ventilator except in the last experiment. All trials were conducted three times with each ventilator, and mean values are reported.

*Suppliers are identified in the Product Sources section at the end of the text.

Determining Factors Affecting Volume

Flow. For all ventilators, the test lung's low lung-compliance setting (1.0 mL/cm H_2O [0.01 L/kPa]) was selected, and the lung simulator was connected to the ventilator circuit. For all ventilators except the Siemen's Servo 900C, the ventilator flowrate was set at 20 L/min, the ventilator rate at 30/min, inspiratory time at 0.66 s, PEEP at 0, and the pressure limit adjusted to produce a peak inspiratory pressure (PIP) of 30 cm H_2O [2.94 kPa]. Tidal volume and end-expiratory volume were measured and recorded. Then, with no change in ventilator settings, the compliance of the simulator was changed to 3.0 mL/cm H_2O [0.03 L/kPa], and pressures and volumes were again recorded.

The procedure was repeated with the ventilator flowrate set at the minimum flow required to reach a PIP of 30 cm H_2O [2.94 kPa] (that is, the PIP was determined only by the ventilator flow, not by the pressure-limit control).

Inspiratory Time. The described procedures were repeated with the ventilator rate at 120/min, and the inspiratory time at 0.17 s (I:E at 1:2), at both the 20 L/min and minimum flow settings.

Ventilator Brand and Mode. To determine the effect of changes in ventilator brand or mode, all of the procedures were repeated with each of the ventilators. In addition, certain modifications or additions to the procedures were made. Sechrist ventilator—procedures were conducted with the ventilator pressure-waveform control adjusted first to deliver a sine wave, and then to produce a square wave. The 335 exhalation block was used for all trials with the Sechrist. InfantStar—procedures were conducted with the ventilator set first for demand flow and then for continuous flow. Siemens Servo 900C—because the Servo 900C has no directly adjustable flowrate control, the pressure control mode was used when the protocol required 20 L/min flow. This mode produces high flows and square inspiratory pressure waves. To achieve minimum flows, the volume mode was used. Minute volume was set at the lowest value required to achieve a PIP of 30 cm H_2O [2.94 kPa]. In both modes, the ventilator's flow waveform control was set to deliver a square flow pattern.

Table 1. Effect of Flow and Inspiratory Time on Maximum Lung Volumes (Test-Lung 'FRC' Change + Tidal Volume) with an Increase in Test-Lung Compliance

		Test-Lung Compliance			
		Baseline 1 mL/cm H_2O	3 mL/cm H_2O	Baseline 1 mL/cm H_2O	3 mL/cm H_2O
Ventilator*	Inspiratory Time (seconds)	Volume with High Flow		Volume with Low Flow	
Servo	0.66	30	82	30	36
	0.17	30	47	29	33
Cub	0.66	30	93	30	38
	0.17	30	43	29	41
BP-200	0.66	30	86	29	43
	0.17	29	50	28	39
InfantStar					
Demand flow	0.66	30	87	30	36
	0.17	30	47	30	39
Continuous flow	0.66	30	86	30	36
	0.17	30	50	30	36
Sechrist					
Square wave	0.66	30	90	30	39
	0.17	29	42	28	36
Sine wave	0.66	30	81	30	45
	0.17	30	60	27	59

* Volumes are in mL; peak inspiratory pressures were set to 30 cm H_2O, with rate of 30 breaths/minute for 0.66-s inspiratory time and rate of 120 for

Ventilator Compliance. Finally, these procedures were repeated with the Cub and BP-200 with the ventilator-circuit compliance increased (by the addition of a large volume humidifier) to 1.5 mL/cm H_2O [0.015 L/kPa].

Results

Effects on MaxV Increases

MaxV invariably increased as lung compliance increased, regardless of ventilator settings, brand, mode, or circuit compliance. The magnitude of MaxV increases was influenced by flow, inspiratory time, brand, and mode.

Flow. For all ventilators tested, MaxV increased by the greatest amount when 20 L/min flows (or the pressure-control mode on the Siemens) were used Table 1, Fig. 1). The mean (SD) MaxV for all ventilators after compliance increased was 86 (4) mL, an increase of 56 mL in the MaxV over MaxV when compliance was at the lower setting. In contrast, when a minimum flow was used, MaxV increased by 39 (4) mL, an increase of 10 mL over mean MaxV at the lower compliance.

Inspiratory Time. Reduction of inspiratory time reduced the degree of increase in MaxV. When flow was high, and inspiratory time was at 0.17 s, the mean increase was 48 (6) mL—just over half

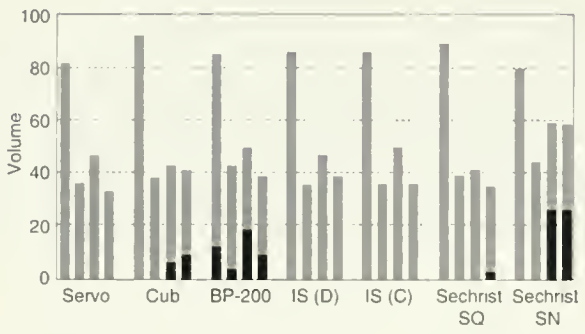

Fig. 1. Maximum lung volume, or MaxV ■ (end-expiratory volume, if any, ■ + tidal volume) after increasing lung compliance from 1 to 3 mL/cm H_2O, with peak inspiratory pressure = 30 cm H_2O, rate = 30 breaths/min for 0.66-s inspiratory time (t_i), and at 120 for 0.17-s t_i. First bar in each group represents 0.66-s t_i and 20 L/min (high flow); second bar represents 0.66-s t_i and low flow (see text); third bar = 0.17-s t_i, high flow; and fourth bar = 0.17-s t_i, low flow. The infantStar (IS) was used in the continuous (C) and demand (D) flow modes, and the Sechrist was used with both the square-wave (SQ) and the sine-wave (SN) flow patterns.

the increase seen when inspiratory time was 0.66 s. When flow was at the minimum setting and the inspiratory time was reduced to 0.17 s, the increase in MaxV was 40 (9) mL.

Ventilator Brand and Mode. Lowest MaxV was always produced with low-flow settings, regardless of ventilator brand. However, short inspiratory time was not always associated with the lowest MaxV for all ventilators. The Siemen's Servo 900C, BP-200, and the Sechrist (square-wave pressure pattern) produced their lowest MaxV at the low-flow, short-inspiratory-time setting. The Cub, Sechrist (sine-wave pressure pattern), and InfantStar (demand flow) produced their lowest MaxV with the low-flow, long-inspiratory-time setting. When used in the continuous-flow setting, the InfantStar produced equally low MaxV at the low-flow settings, regardless of the inspiratory time.

Ventilators varied substantially in their ability to minimize MaxV increases through manipulation of ventilator settings. The Siemens Servo 900C and the InfantStar (demand or continuous flow) had increases of less than 6 mL in MaxV with certain ventilator settings, whereas the BP-200, Cub, and Sechrist (both sine- and square-wave pressure patterns) produced increases of at least 8 mL or more at all settings.

MaxV increases were generally due to increases in tidal volume. Only the BP-200 and the Sechrist (sine-wave pressure pattern) had end-expiratory lung volumes of more than 6 mL after compliance increased (Fig. 1).

Circuit Compliance. When ventilator-circuit compliance was high, MaxV increases secondary to lung compliance increases were substantially higher than when ventilator compliance was low, for all combinations of settings except the high-flow, 0.66-s inspiratory time (Fig. 2). At the 120-breaths/min setting, it was not possible to reach the PIP with a flow of less than 20 L/min; therefore, only the high-flow setting is reported at 0.17-s inspiratory time.

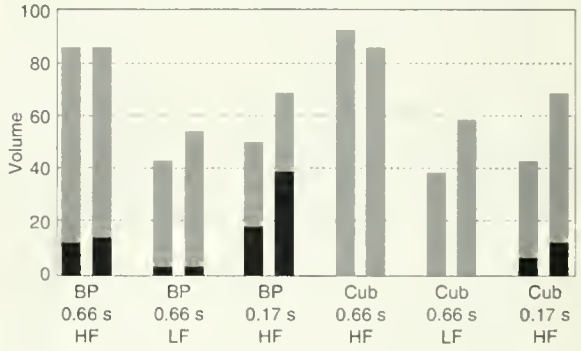

Fig. 2. End-expiratory ■ and tidal volumes ■ produced when test-lung compliance was increased and ventilator-circuit compliance was 0.2 mL/cm H_2O (first of each pair of bars) or 1.5 mL/cm H_2O (second of each pair). Ventilators and ventilator settings were BP-200 (BP) and Cub with inspiratory time (t_i) at either 0.17-s or 0.66-s, at high (HF) 20 L/min, or low (LF) flows (see text). At 0.17-s t_i, minimum and maximum flow were the same. Maximum lung volume, or MaxV = end-expiratory volume (if any) + tidal volume.

Effects on Tidal Volume

Ventilator Brand, Mode, and Settings. Tidal volume changes at the low-compliance setting varied little due to changes in inspiratory time or flow (Table 2). Using the tidal volume measured at the 0.66-s inspiratory time, high-flow setting as a reference, tidal volume changed by more than 10% (3 mL) only with the BP-200 and Sechrist (sine-wave pressure pattern) due to decreasing flow and/or de-

Table 2. Effect of Flowrate on Tidal Volume with Long and Short Inspiratory Times and Test-Lung Compliance Constant at 1 mL/cm H_2O

Ventilator*	Inspiratory Time (seconds)	Tidal Volume: High Flow	Tidal Volume: Low Flow
Servo	0.66	30	30
	0.17	30	29
Cub	0.66	30	30
	0.17	28	29
BP-200	0.66	26	29
	0.17	26	25
InfantStar			
Demand flow	0.66	30	30
	0.17	30	30
Continuous flow	0.66	30	30
	0.17	30	30
Sechrist			
Square wave	0.66	30	30
	0.17	29	28
Sine wave	0.66	30	30
	0.17	26	27

* All volumes are in mL. Peak inspiratory pressure = 30 cm H_2O; PEEP = 0; rate = 30/min at 0.66-s inspiratory time, 120/min at 0.17-s inspiratory time.

creasing inspiratory time. Of interest, decreasing flow resulted in an increase in tidal volume with the BP-200. Reduction in inspiratory time produced a greater than 10% drop in tidal volume at both flowrates with the Sechrist (sine-wave pressure pattern), and a similar loss in tidal volume with the BP-200 only at the low-flow settings.

Circuit Compliance. With the BP-200, a high ventilator-circuit compliance magnified the effect of reducing inspiratory time on changes in tidal volume when lung compliance was 0.1 mL/cm H_2O. Tidal volume dropped 28% with the BP-200 when inspiratory time was decreased.

Effect on Peak Inspiratory Pressure

PIP dropped when compliance improved. The least change was noted in the long-inspiratory-time, high-flow settings (0-2 cm H_2O [0-0.196 kPa]). Decreases in PIP with the other settings were as great as 14 cm H_2O [1.37 kPa].

Discussion

Lung overdistention may increase the risk for pulmonary air leak,[8] reduce effective ventilation,[9] and unfavorably alter intracranial pressures and blood flow.[10-12] My results suggest that lung volume increases secondary to improved compliance are influenced by ventilator brand and ventilator settings. Reduction in inspiratory time or flow can reduce the extent of lung overdistention, but the exact combination that produces the lowest MaxV (ie, the change that could produce overdistention) varies among ventilators.

Reduction in inspiratory time has been shown by several investigators to reduce the risk of pulmonary air leak.[13,14] The mechanism by which pulmonary air leak is reduced is unclear. However, this study and others suggest that inspiratory time may affect the risk of pneumothorax by limiting the increase in lung volume associated with increasing lung compliance.

Previously published reports demonstrating a reduced risk for pulmonary barotrauma with short inspiratory times used ventilators not commonly in use today (eg, the Babybird[13,14]). This study confirms, with a more contemporary group of ventilators, that short inspiratory times reduce the amount of lung volume increase as lung compliance improves. However, the role of flow in the prevention of barotrauma has not been fully explored. For several of the ventilators tested, the potential for lung overdistention was lowest at long inspiratory times, when minimum flows were used. In some cases, lung volume changes were substantially greater when short inspiratory times were used, in contrast to long-inspiratory-time, low-flow settings. This suggests that clinical studies concerning the efficacy and safety of various ventilatory strategies should not be generalized beyond the ventilator brands used.

It is not clear what accounts for the differences between ventilators in the response to increasing lung compliance, although exhalation valve design probably accounts for some of the differences observed. For example, the Sechrist ventilator relies on its exhalation valve to produce the pressure waveform selected by the operator. When a square wave is selected, the exhalation valve closes rapidly, allowing for a more rapid increase in ventilator pressure. In contrast, when the sine wave is selected, the valve closes slowly, allowing for a more gradual buildup of pressure. The exhalation valve also opens slowly when the sine wave is selected, accounting for the large end-expiratory volumes seen.

If lung overdistention is to be minimized during recovery from RDS, peak airway pressures must be allowed to drop spontaneously as lung compliance improves. Use of high flows prevents this. When flows greatly in excess of the minimum needed to achieve the desired PIP are used, the ventilator will be able to maintain the preset pressure even in the face of increasing lung compliance. However, when minimum flows are used, ventilator pressures cannot be maintained because the volume necessary to accommodate the increased lung compliance is not available. The pressure developed within the circuit drops, and tidal volume increases are minimized. In other words, the lower the flow set on the ventilator, the more a continuous-flow, time-cycled ventilator acts as a volume-limited (or flow-limited) ventilator. Despite being commonly referred to as pressure ventilators, time-cycled continuous-flow ventilators can, if no pressure limit is employed, deliver a constant tidal volume (ie, be volume- or flow-limited). When it is operated in the flow- or volume-limited mode, the ventilator's peak inspiratory pressure and pressure waveform are dependent on patient compliance and resistance.[15]

In contrast, when the ventilator is operated as a pressure-controller, the pressure waveform and peak inspiratory pressure are (ideally) not affected by patient compliance or resistance.[15] To allow the ventilator to function as a pressure-controller, despite large changes in resistance or compliance, high flows are needed. As preset flow is reduced from the maximum toward the minimum that allows the ventilator to meet the desired PIP, the ventilator responds more and more like a flow- or volume-controller as compliance increases, despite continuing to act as a pressure-controller should compliance decrease.

Most of the trials in this study were conducted with an unusually low circuit compliance of 0.2 mL/cm H_2O. This was done in order to highlight differences between ventilator brands and eliminate any extraneous influence from the ventilator circuit. However, a greater increase in MaxV was noted in the two ventilators tested with a high-compliance circuit. The degree to which ventilator pressure drops is determined not by the increase in patient compliance alone but by the increase in the sum of ventilator and lung compliance. If lung compliance is low in relation to circuit compliance, a large increase in lung compliance has little effect on the sum of patient and ventilator compliance. The drop in ventilator peak inspiratory pressure is minimal, and, hence, tidal volume increases substantially. Conversely, when lung compliance is high in relation to ventilator-circuit compliance, then a large increase in lung compliance has a proportionately larger effect on the sum of the two. The decrease in ventilator peak pressure is greater, and tidal volume increases are minimized.

MaxV may also increase due to increases in end-expiratory volume when a high-compliance ventilator circuit is used. Scott et al[16] demonstrated that ventilator-circuit compliance can cause a large increase in auto-PEEP. They studied an adult volume ventilator (MA-1). This study extends their findings to infant ventilators.

When ventilator-circuit compliance is high, more gas is contained in the circuit. This gas, when combined with the patient's exhaled gas, increases the total amount of gas that must be emptied from the circuit in the expiratory time allotted. Hence, exhalation may not be complete, and end-expiratory volumes may increase.

Spontaneously decreasing peak airway pressures associated with improving lung compliance can easily be interpreted as increasing leak around the endotracheal tube, leading practitioners to adjust pressures back to their original level. More widespread use of tidal volume monitors should allow clinicians to differentiate true changes in patient compliance from other causes of ventilator pressure variability.

The relative importance of lung overdistention in the etiology of pneumothorax during periods of increasing compliance, as in the recovery phase of RDS, has not been established. In fact, many studies of surfactant replacement therapy have demonstrated a reduced risk for pulmonary air leak,[17] suggesting that improvements in lung compliance protect against pulmonary air leak, despite associated increases in lung volume. Other mechanisms (such as reduction in asynchronous breathing[18]) have been proposed to account for the reduced risk for air leak when short inspiratory times are used. However, another potential benefit of reducing flow and inspiratory time to minimize lung overdistention is suggested by the observations of several investigators that increased intrapulmonary pressures unfavorably alter intracranial pressure and blood flow, especially during periods of increasing lung compliance or during asynchronous breathing.[10-12,19]

Although several ventilators tested produced lower MaxV with the long-inspiratory-time, low-flow setting, reduction in inspiratory time may be the preferred method of ventilation because it has been shown previously that shortened inspiratory times reduce the amount of asynchronous breathing.[18] However, the long inspiratory time selected for my study is closer to the short inspiratory time shown to be beneficial in the study by Heicher et al.[13]

My study is in general agreement with previously published reports by Greenough and Greenall[20] and Boros et al[21] on tidal volume changes at high ventilator rates. Two of the ventilators tested in my study were also studied by Boros et al (BP-200 and Sechrist). Boros et al found a greater reduction in tidal volume with increasing rate (more than 50% as rate increased from 25 to 120). However, their lung model had a time constant twice that of mine, and the ventilator-circuit compliance was nearly four times greater. Both factors increase tidal volume loss due to shortened inspiratory times.

Greenough and Greenall's study included three ventilators (Sechrist, Cub, and BP-200) also tested in this study. They used a lung model with a time constant identical to mine, but information is not provided on circuit compliance. Both their study and mine showed tidal volume losses of less than 10% secondary to decreased inspiratory time for the Cub and Sechrist. However, their results demonstrated a 29% loss in volume with the BP-200, consistent with my findings when ventilator-circuit compliance was high.

Studies of ventilator performance using lung simulators have obvious drawbacks, and some caution is warranted prior to clinical application of results. Chief among these drawbacks is the fact that almost all lung models consist of a single, rigid chamber to simulate the alveoli (compliance) and a single narrow tube or connector to simulate the airways (resistance). In reality, the lung consists of thousands of units each with its own time constant. Lung units with very high and very low time constants can coexist in the lung, and a lung model with a single time constant cannot completely reflect the lung's response to mechanical ventilation.

Conclusions

In conclusion, lung overdistention can be minimized through reduced flows and inspiratory times and the use of low-compliance ventilator circuits. The clinically important variations in potential lung overdistention produced by different ventilators suggest that studies of the effects of inspiratory time be considered specific to the brand tested.

PRODUCT SOURCES

Ventilators:

Siemens Servo 900C, Siemens Life Support Systems, Schaumberg IL

Bourns BP-200 and Bear Cub, Bear Medical Systems, Riverside CA

Sechrist IV 100B, Sechrist Industries, Anaheim CA

InfantStar, Infrasonics Industries, San Diego CA

Ventilator Tester:

Bio-Tek VT-2, Bio-Tek Instruments Inc, Winooski VT

REFERENCES

1. Simbruner G. Inadvertent positive end-expiratory pressure in mechanically ventilated newborn infants: detection and effect on lung mechanics and gas exchange. J Pediatr 1986;108:589-595.
2. Hird M, Greenough A, Gamsu H. Gas trapping during high frequency positive pressure ventilation using conventional ventilators. Early Human Dev 1990;22:51-56.

3. Bhat R, Dziedzic K, Bhutani VK, Vidyasagar D. Effect of single dose surfactant on pulmonary function. Crit Care Med 1990;18:590-595.
4. Kao LC, Durand DJ, Phillips BL, Nickerson BG. Oral theophylline and diuretics improve pulmonary mechanics in infants with bronchopulmonary dysplasia. J Pediatr 1987;111:439-444.
5. Carlo WA, Beoglos A, Siner BS, Martin RJ. Neck and body position effects on pulmonary mechanics in infants. Pediatrics 1989;84:670-674.
6. Alfredson T, Ogata E. Simulated tidal volume, lung pressure, and mean airway pressure using short and long inspiratory time mechanical ventilation (abstract). Respir Care 1981;26:1130.
7. Lewis RM, Thompson SL, Goldberg AI. Respiratory care for the infant and child. In: Burton GG, Hodgkin JE, Ward JJ, eds. Respiratory care: a guide to clinical practice, 3rd ed. Philadelphia: JB Lippincott Co, 1991: 757-820.
8. Greenough A, Dixon AK, Robertson NRC. Pulmonary interstitial emphysema. Arch Dis Child 1984;59:1046-1051.
9. Fisher JB, Mammel MC, Coleman JM, Bing DR, Boros SJ. Identifying lung overdistension during mechanical ventilation by using volume-pressure loops. Pediatr Pulmonol 1988;5:10-14.
10. Leahy FA, Durand M, Cates D, Chernick V. Cranial blood volume changes during mechanical ventilation and spontaneous breathing in newborn infants. J Pediatr 1982;101:984-987.
11. Milligan DW. Positive pressure ventilation and cranial volume in newborn infants. Arch Dis Child 1981;56: 331-335.
12. Cowan F, Thoresen M. The effects of intermittent positive pressure ventilation on cerebral arterial and venous blood velocities in the newborn infant. Acta Paediatr Scand 1987;76:239-247.
13. Heicher DA, Kasting DS, Harrod JR. Prospective clinical comparison of two methods for mechanical ventilation of neonates: rapid rate and short inspiratory time versus slow rate and long inspiratory time. J Pediatr 1981;98:957-961.
14. Bland RD, Kim MH, Light MJ, Woodson JL. High frequency mechanical ventilation in severe hyaline membrane disease: an alternative treatment? Crit Care Med 1980;8:275-280.
15. Chatburn RL. A new system for understanding mechanical ventilators. Respir Care 1991;36:1123-1155.
16. Scott LA, Benson MS, Pierson DJ. Effect of inspiratory flowrate and circuit compressible volume on auto-PEEP during mechanical ventilation. Respir Care 1986;31: 1075-1079.
17. Yee WF, Scarpelli EM. Surfactant replacement therapy. Pediatr Pulmonol 1991;11:65-80.
18. Greenough A, Wood S, Morley CJ, Davis JD. Pancuronium prevents pneumothoraces in ventilated premature babies who actively expire against positive pressure ventilation. Lancet 1984;1:1-4.
19. Rennie JM, South M, Motley CJ. Cerebral blood flow velocity variability in infants receiving assisted ventilation. Arch Dis Child 1987;62:1247-1251.
20. Greenough A, Greenall F. Performance of ventilators at fast rates commonly used in neonatal intensive care units. Pediatr Pulmonol 1987;3:357-361.
21. Boros SJ, Bing DR, Mammel MC, Hagen E, Gordon MJ. Using conventional infant ventilators at unconventional rates. Pediatrics 1984;74:487-492.

Rapid Analysis of Exhaled CO_2 To Assess Endotracheal Tube Placement

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BACKGROUND: Assessing proper placement of an endotracheal tube can be difficult. Assessment techniques include visualization of the tube in the larynx, auscultation of the chest and abdomen, assessment of compliance by the 'feel' of the resuscitation bag, assessment of radiographs, and observations related to the presence or absence of (1) humidity in the tube, (2) patient phonation, or (3) gastric contents in the tube. Despite these techniques, tubes can be misplaced. We reasoned as have others that the presence or absence of CO_2 might be a more rapid and specific indicator of tube placement. METHOD & MATERIALS: All endotracheal intubation attempts by Respiratory Care Services at Duke University Medical Center from July 1989 to October 1990 were studied. A Biochem 515 portable CO_2 monitor was used to detect CO_2 in the gas returned through the endotracheal tube. Proper tube position was ultimately confirmed by chest radiograph. RESULTS: We analyzed the 933 patient intubations that occurred during the study period, and found that in 915/925 successful intubations CO_2 was present (true positive 99%), and in 8/8 unsuccessful intubations CO_2 was absent (true negative 100%). No unsuccessful intubations had CO_2 present (false positive 0%) and 10/925 successful intubations had CO_2 absent (false negative of 1.1%). Of the 10 successful intubations that did not result in endotracheal CO_2, 7 subjects were believed to have had little or no CO_2 in the lung due to poor cardiopulmonary perfusion, 1 was associated with a faulty CO_2 analyzer, and for 2 there was no explanation for the absence of CO_2. CONCLUSION: Our results confirm the earlier work of Owen and Cheney reported in this journal. Although no one sign is completely conclusive for verification of endotracheal tube placement, CO_2 detection is quick and easy, and the presence of CO_2 confirms proper intubation. (Respir Care 1992;37:1161-1165.)

Introduction

Endotracheal intubation is a life-saving procedure routinely performed by physicians, respiratory care practitioners, certified registered nurse anesthetists, and paramedics. Assessing proper placement of the endotracheal tube (ETT) can be difficult. Standard techniques include visualization of the tube in the larynx; auscultation of the chest and abdomen; assessment of compliance via the 'feel' of the resuscitation bag; radiologic assessment; and observations related to the presence or absence of (1) humidity in the tube, (2) patient phonation, or (3) gastric contents in the tube. Despite these techniques, up to 1.8% of endotracheal intubations assessed to be correct in fact are misplaced in the esophagus.[1,2]

A more specific approach to ETT placement has been the measurement of carbon dioxide (CO_2) in the tube.[3-6] The premise has been that if CO_2 is present, the tube is in the trachea, and if CO_2 is absent, the tube is in the esophagus. This premise is based on the presumption that the lung releases gas

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containing substantial CO_2, whereas the gastrointestinal tract does not. However, this may not always be true. In severe lung hypoperfusion, tracheal CO_2 may be very low[7] and, conversely, following ingestion of large amounts of CO_2 (eg, carbonated beverages, antacids) stomach gas can contain CO_2.[8] We thus assessed a large number of intubation attempts in our hospital with immediate CO_2 analysis of ETT gas to establish the accuracy and reliability of this technique for determining proper tube placement.

Method and Materials

All endotracheal intubation attempts by Respiratory Care Services at Duke University Medical Center from July 1989 to October 1990 were studied. In our hospital, Respiratory Care Services is the primary responder for all emergency and elective endotracheal intubations. For all attempts at intubation, we documented indications, number of attempts, route (nasal vs oral), results of postintubation auscultation and observation for evidence of respiratory compromise, and radiologic assessment. In addition, during this period we analyzed all gas returned from ETT using a portable CO_2 analyzer (Biochem 515, Biochem Corp, Waukesha WI) to determine the presence or absence of CO_2 . The analyzer uses infrared absorption to detect CO_2. Its qualitative response is displayed on a bank of 8 light-emitting diodes (LEDs). We decided prospectively to accept a strong signal (the illumination of > 3 LEDs) as evidence of the presence of CO_2. The analyzer requires no warmup or calibration and has a response time of 160 milliseconds. Exhaled gas was analyzed for 8-10 breaths following intubation. Patients were also assessed for ETT placement with the usual clinical evaluation and chest radiograph. If there was any clinical concern that the tube was in the esophagus, the tube was immediately removed and reinserted.

Data were analyzed in the following way. The true-positive condition was quantitated as the number of times CO_2 was detected/number of times the ETT was determined to be placed properly by subsequent auscultation, arterial blood gas analysis, and chest radiograph. The true-negative condition was quantitated as the number of times CO_2 was not detected immediately postintubation/number of times the ETT was determined by auscultation, arterial blood gas analysis, and chest radiograph to be in esophagus. The false-positive condition was quantitated as the number of times CO_2 was detected/number of times the ETT was placed incorrectly. The false-negative condition was quantitated as the number of times CO_2 was absent/number of times the ETT was placed correctly.

Table 1. Intubation Attempts by Respiratory Care Services at Duke University Medical Center from July 1989 to October 1990

Total number of patients intubated	977
Route	
Nasal	88
Oral	889
Indication	
Airway protection	83
Secretion clearance	10
Ventilatory support	614
Cardiopulmonary arrest	266
Airway obstruction	4
Total patients with complete data for analysis	933

Results

During the 16-month evaluation period, 977 patients were endotracheally intubated by the Duke University Respiratory Care Services, and 933 of these patients had complete data for analysis (Table 1). True positives and true negatives for proper endotracheal tube placement by CO_2 analysis were 99% and 100%, respectively. False-positive and false-negative results for successful intubation by CO_2 analysis were 0% and 1.1%, respectively (Fig. 1). In the 10 subjects successfully intubated from

$$\textit{true positive} = \frac{\textit{CO}_2\ \textit{present}}{\textit{successful intubation}} = \frac{915}{925} = 99\%.$$

$$\textit{false positive} = \frac{\textit{CO}_2\ \textit{present}}{\textit{unsuccessful intubation}} = \frac{0}{8} = 0\%.$$

$$\textit{true negative} = \frac{\textit{no CO}_2\ \textit{present}}{\textit{unsuccessful intubation}} = \frac{8}{8} = 100\%.$$

$$\textit{false negative} = \frac{\textit{no CO}_2\ \textit{present}}{\textit{successful intubation}} = \frac{10}{925} = 1.1\%.$$

Fig. 1. Results of CO_2 analysis in 933 intubation attempts by Respiratory Care Services at Duke University Medical Center from July 1989 to October 1990.

whom no ETT CO_2 was recovered, 5 were undergoing cardiopulmonary resuscitation (CPR) and were believed to have poor pulmonary perfusion, and 2 were found at autopsy to have massive pulmonary emboli. The CO_2 analyzer used on 1 patient was subsequently found to be faulty, and in 2 patients no apparent explanation was found for the absence of CO_2 in the properly placed ETT.

Discussion

The placement of the ETT should be verified quickly and accurately following attempted endotracheal intubation (whether by oral or nasal route). Esophageal intubation is the most avoidable early complication of intubation (leading in the categories of death, neurologic damage, and anesthesia mortality).[1] Indicators of proper placement include[2]

- Seeing the endotracheal tube pass through the glottis unimpeded—many clinical conditions and differences in tracheal anatomy prevent the operator from clearly making this observation. (This concept also assumes that the tube does not become dislodged after placement is confirmed visually.)

- Auscultating breath sounds bilaterally after intubation to verify air movement when the patient receives a mandatory breath—breath sounds should be auscultated at the mid-axillary line to ensure that they are audible and occur with each manual insufflation of the bag. In certain patient conditions (obesity, CPR delivery, noisy environments such as in an ambulance, presence of the drapes of a sterile chest field), it may be impossible to verify tube placement by auscultation.

- Observing symmetrical chest excursion with the delivery of a mandatory breath—several clinical conditions may impede this observation (eg, obesity, pendulous breasts, or a fixed chest wall as seen in the patient with COPD).

- Auscultating the epigastrium and observing the stomach—breath sounds noted more loudly over the umbilicus and stomach bloating with manual ventilation may indicate that the tube has been placed in the esophagus.

- Noting the feel of the manual resuscitation bag—the feel through an ETT placed in the trachea should be different from the feel through a tube in the esophagus because the lungs are more compliant than the esophagus.

- Hearing vocal sounds from the patient after intubation—the patient who speaks, coughs, or makes any tracheal sounds after intubation probably is not intubated in the trachea. No vocal sounds should be possible if the tube has been passed through the glottis.

- Free movement of air through the ETT of spontaneously breathing patients—if air movement into and out of the ETT is easy to feel and hear, the tube is almost certainly in the trachea (assuming the patient can take spontaneous breaths).

- Gastric contents expelled from the ETT (usually a sign of esophageal intubation)—unless there has been recent gastric aspiration, no dark, bile-like fluid (as seen in the stomach) is seen in the airways. For patients who are experiencing pulmonary edema, fluid in the lungs may range from yellow to pink tinged and will usually be a much smaller amount than noted from the stomach.

- Condensation present within the ETT when the tube is in the trachea—because the air in the lungs is heated and humidified by normal physiologic processes, moisture usually condenses in exhaled air. This method is not foolproof, however, because gastric gas can mimic this phenomenon in esophageally intubated patients.

- Rapidly developing physiologic instability when the tube is placed in the esophagus (especially in infants and neonates)—bradycardia and desaturation occur rapidly in infants and neonates, and these are very likely indicative of esophageal intubation. Most patients respond very quickly to extubation and hyperoxygenation/manual ventilation if the tube is mistakenly

placed in the esophagus. Adults will also show clinical deterioration if the tube is mistakenly placed in the esophagus, but this may occur more slowly.[9]

- Radiographic confirmation of tube placement—most ETTs are fitted with a radiopaque strip that can be seen on the chest radiograph. Tracheal cannulation, main-stem intubation, and tube distance from the carina can all be determined from the postintubation chest radiograph.

- Direct revisualization by laryngoscopy—if the previously described means of verifying tube placement fail and some question still exists, direct visualization may be necessary. This requires another laryngoscopy to see whether the tube has passed into the trachea or into the esophagus. Again, this method may be difficult if the patient's clinical condition makes manipulation of the neck difficult or dangerous (eg, possible cervical spine damage, presence of a cervical collar, cervical arthritis). In addition, variations in the laryngeal anatomy can make revisualization difficult and sometimes unreliable. Visualization of the trachea allows an accurate determination to be made.

- Pulse oximetry—pulse oximetry during intubation may reveal esophageal intubation. As noted earlier, hemoglobin desaturation occurs more quickly in the infant or neonate than in adults. Nevertheless, hemoglobin desaturation occurring in adults can be a sign of esophageal intubation.

- Use of an introducer—a so-called introducer, a 60-cm graduated plastic catheter, when inserted through the tracheally-placed ETT, meets resistance at about the 28- to 32-cm mark. If the tube is placed in the esophagus, no resistance is met as the introducer passes unopposed into the stomach.

Another CO_2 analyzer suitable for use during intubation is the portable, disposable colorimetric device (EasyCap, Nellcor, Hayward CA), which has a reagent sensitive to the presence of exhaled CO_2.[9,10] A color change from the baseline within 6 breaths is considered to be a positive indication of CO_2 and suggests tracheal intubation. This device is limited to a minimal age-weight relationship (8 years of age or 60 pounds) because of the intrinsic dead space (about 38 mL).[11]

Our results demonstrate that, in addition to conventional assessment techniques, CO_2 analysis of ETT gas is a reliable adjunct. These results confirm the work of others[5] and extend these observations to a very large population sample. Of note is that only 10 of 925 successful intubations did not have CO_2 present. Of these 10, 7 were related to low perfusion states and thus caution should be used in interpreting low or absent CO_2 values under such circumstances.[7] On the other hand, no cases of false-positive CO_2 measurements were recorded in any of the 8 unsuccessful intubations. Concern has been raised that ingestion of large amounts of CO_2-containing substance (eg, carbonated beverages) could produce enough CO_2 in gastrointestinal gas to give a false-positive result for ETT placement.[8] We did not observe this and believe that our policy of requiring a consistently strong CO_2 signal over an 8-10 breath interval minimizes this possibility.

Conclusion

Our results confirm the earlier work of Owen and Cheney reported in this journal. Although no one sign is completely conclusive for verification of endotracheal tube placement, CO_2 detection is quick and easy and the presence of CO_2 confirms proper intubation.

REFERENCES

1. Stewart RD, Paris PM, Winter PM, Pelton GH, Cannon GM. Field endotracheal intubation by paramedical personnel: success rates and complication. Chest 1984;85: 341-345.
2. Birmingham PK, Cheney FW, Ward RJ. Esophageal intubation: a review of detection techniques. Anesth Analg 1986;65:886-891.
3. Murray IV, Modell JH. Early detection of endotracheal tube accidents by monitoring carbon dioxide concentration in respiratory gas. Anesthesiology 1983;59:344-346.
4. Linko K, Palohermo M, Tammisto T. Capnography for detection of accidental esophageal intubation. Acta Anaesthesiol Scand 1983;27:199-202.

5. Owen RL, Cheney FW Jr. Use of an apnea monitor to verify endotracheal intubation. Respir Care 1985;30: 974-976.
6. Anton WR, Gordon RW, Jordan TM, Posner KL, Cheney FW. A disposable end-tidal CO_2 detector to verify endotracheal intubation. Ann Emerg Med 1992;20:82-86.
7. Fulk JL, Rackow EC, Weil MH. End-tidal carbon dioxide concentration during cardiopulmonary resuscitation. N Engl J Med 1988;318:607-611.
8. Sum Ping ST, Mehta MP, Symreng T. Reliability of capnography in identifying esophageal intubation with carbonated beverage or antacid in the stomach. Anesth Analg 1991;73:333-337.
9. Gorback MS. Endotracheal intubation and airway management. In: Fulkerson WJ, MacIntyyre NR, eds. Complications of mechanical ventilation. Probl Respir Care 1991;4(1):1-24.
10. Strunin L, Williams T. The FEF end tidal carbon dioxide detector (letter). Anesthesiology 1989;71:621-622.
11. Jones BR, Dorsey MJ. Disposable end tidal CO_2 detector: minimal CO_2 requirements (abstract). Anesthesiology 1989;71:359.

The Effect of Partial Upper-Airway Bypass on Subglottic Pressure during Acute Lung Injury in Sheep

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BACKGROUND: Upper-airway function is thought to influence gas exchange and lung volume through an expiratory braking mechanism producing increased subglottic pressures. We designed an experiment to examine the effect of partial upper-airway bypass on the development of subglottic pressure during acute lung injury (ALI). MATERIALS & METHODS: Six healthy adult domestic sheep underwent surgical placement of a tracheal Kistner button in the upper trachea and carotid transposition for arterial blood access. On the following day, baseline airway-pressure tracings were obtained at the Kistner button, and respiratory airflow tracings were obtained at the mouth. All measurements and recordings were initially made with the Kistner buttons closed to atmosphere, allowing respiration only across the upper airway, and then were repeated with the Kistner buttons open, allowing partial respiratory bypass of the upper airway. Twenty-four hours following the baseline recordings, an intravenous infusion of oleic acid (0.08 mL/kg) was administered to produce ALI. On the following day, the recordings were repeated and arterial blood gas samples were obtained. RESULTS: Subglottic pressures at baseline with the Kistner buttons closed (3.5 cm H_2O [0.34 kPa]) were significantly larger than when the Kistner buttons were open, (1.4 cm H_2O [0.14 kPa], $p < 0.0001$). The respiratory rates, inspiratory times (t_i), expiratory times (t_e), and the t_i-to-t_e ratio (I:E) were unchanged with the Kistner buttons open to atmosphere. With ALI, subglottic pressure was significantly increased ($p > 0.0001$), compared to baseline (12.3 cm H_2O [1.21 kPa] vs cm H_2O [0.34 kPa], respectively). Other variables demonstrated a decrease in the t_i ($p < 0.001$) and a decrease in the I:E ($p < 0.0001$) compared to baseline measurements. No significant difference existed in t_e or the respiratory rates. Opening of the Kistner buttons during acute lung injury produced a significant decrease in subglottic pressures ($p < 0.0001$) and t_e ($p = 0.0031$), whereas the t_i and I:E significantly increased with this maneuver ($p = 0.0002$ and $p < 0.0001$, respectively). The partial pressures of oxygen were found to be significantly greater ($p = 0.0401$) with the Kistner buttons closed to atmosphere (64.3 torr [8.58 kPa]) than when they were open (53.5 torr [7.13 kPa]), during ALI. CONCLUSIONS: Our results suggest that (1) partial bypass of the upper airway may be associated with a decrease in the development of subglottic pressures, and (2) respiratory timing and pulmonary gas exchange are influenced by the upper airway during ALI. (Respir Care 1992;37:1166-1174.)

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Introduction

The upper airway is thought to serve as an expiratory retard that, when bypassed, may impair gas exchange and decrease lung volumes.[1] Several clinical studies in adults and infants have attempted to investigate the influence of upper-airway bypass on gas exchange and lung volumes.[2-8] A criticism of several of these studies has been the presence of endotracheal intubation, which can lead to reflexive increases in pulmonary airways resistance, producing secondary changes in lung volumes and gas exchange.[9-11] We developed an animal model without tracheal intubation to investigate the impact of partial upper-airway bypass on the development of subglottic pressure during acute lung injury (ALI). Subglottic airway pressure was examined because it normally approximates alveolar pressure and may reflect changes occurring in lung volumes and gas exchange.[12] An ALI model was utilized to test our hypothesis that with ALI, the upper airway plays an important role in the development of positive airway pressures for the maintenance of lung volumes and gas exchange.

Materials and Methods

Preparation of the Animal

Six adult domestic sheep were used for the experimental study. Adult sheep were selected for their size, allowing easy attachment of a Kistner button onto the tracheas of the animals. Additionally, the gross anatomy of the adult sheep's respiratory system is generally similar to that of man, allowing comparisons to be made between the animal model and man. The research was conducted in compliance with the Animal Welfare Act and other federal statutes and regulations relating to animals and experiments involving animals. We adhered to the principles stated in the Guide for the Care and Use of Laboratory Animals (NIH Publication 86-23, 1985). The experimental protocol was approved by the institution's Laboratory Animal Use Committee prior to the beginning of the study.

All sheep underwent routine laboratory screening and quarantine procedures of 30 days' duration to eliminate parasitism and establish homeostasis. No food was provided for 24 hours, and water was withheld for 12 hours prior to the start of the experiment. The sheep were anesthetized with tiletamine hydrochloride 10-15 mg/kg and zolazepam hydrochloride 10-15 mg/kg given intravenously.* The animals were then intubated with a 7- to 9-mm diameter endotracheal tube. They were positioned supine, using support bags on the operating table, and anesthesia was maintained with the inhalational agent isoflurane. The fractional inspired oxygen concentration (F_{IO_2}) was adjusted to maintain arterial oxygen saturations greater than 92%, measured by a pulse oximeter attached to the animal's tongue. The animals were also monitored by electrocardiogram, blood pressure, expiratory end-tidal carbon dioxide, rectal temperature, and by esophageal stethoscope to provide constant auditory evaluation of the animal's heartbeat.

For the carotid transposition, a surgical incision was performed along the lateral aspect of the left external jugular vein overlying the carotid artery. Sharp and blunt dissection were used to isolate the carotid artery from its adjacent structures. The carotid artery was then brought up to a subcutaneous position after the formation of a floor beneath it with the surrounding muscle. The carotid artery transposition was performed to allow easier access to the carotid artery for blood gas sampling. The skin was then closed over the artery. A new surgical incision was then made on the ventral surface of the animal's neck approximating the third and fourth tracheal rings. Sharp dissection between the fascial planes of the sternocephalicus and sternothyroideus muscles allowed exposure of the trachea. A 1% solution of lidocaine was infiltrated in and around the cartilaginous and serosal surfaces of the trachea from rings 2 to 5. An incision was made through a tracheal ring (ring 3 or 4), with excision of the tracheal cartilage forming a stoma with an internal diameter of 6-8 mm, and a Kistner button was placed overlying the stoma. The Kistner buttons were 13.5 mm in outer diameter, 9.4 mm in inner diameter, and 60 mm in length.

The flange of the Kistner button was sewn onto the external serosal surface of the trachea, using six to eight 2-0 silk sutures, which were passed into the

*Commercial suppliers are identified in the Product Sources section at the end of the text.

cartilage of the trachea for added stability and then overlaid with adventitia for an occlusive seal. The attachment of the Kistner button was such that accidental protrusion into the trachea was avoided by the greater size of the Kistner button flange than the size of the stoma and by the method of sewing the flange directly onto the external serosal surface of the trachea. Fascial planes, muscles, and skin were closed around the Kistner button, using appropriate suture. The Kistner buttons were left capped, and the animals were allowed to awaken from the effects of the anesthesia and were then subsequently extubated.

All animals were observed for 24 hours following placement of the Kistner buttons and were offered ad libitum feed and water. Experimental measurements were carried out after this observation period. At the conclusion of the data collection, the animals were humanely euthanized with an intravenous injection of barbiturate.

Measurements

The experimental design required placement of a molded plastic animal anesthesia mask with a soft plastic diaphragm over the sheep's mouth and nose. Care was taken to ensure that an adequate seal was present between the mask and the animal's snout. The mask was attached to a pneumotachograph (Fleisch Model 0, dead space 4.7 mL) for the recording of airflow (Fig. 1). The pneumotachograph was connected by tubing to a pressure transducer, from which the signal was routed to a dual-bridge amplifier. The signals derived from the amplifier were linked to a physiologic waveform recorder.

Airway-pressure tracings were recorded from a Y-connector attached to the open end of the Kistner button, with care taken that a tight seal was present at the connection (Fig. 1). One opening of the Y-connector was capped and the other end was attached by plastic tubing to a pressure transducer, which was linked to the dual-bridge amplifier. The pressure-recording system was zeroed to atmospheric pressure by opening the capped end of the Y-connector during zeroing. The pressure-recording system was calibrated using a water-filled manometer. Zeroing and calibration of the system were performed prior to each animal measurement. The airflow pattern and airway-pressure tracings were recorded simultaneously on the physiologic waveform recorder.

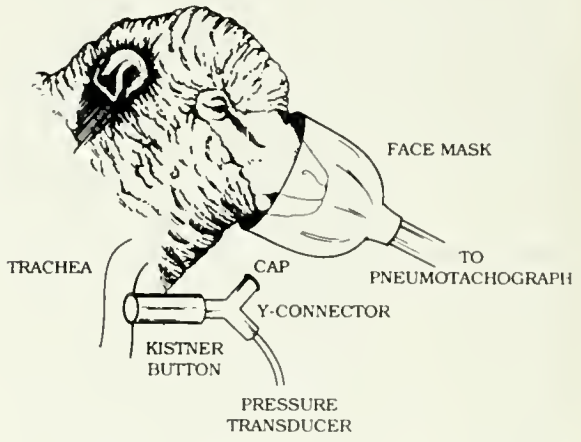

Fig. 1. The sheep model, showing the airway connections with the pneumotachograph and the pressure transducer.

All baseline recordings and measurements were made 24 hours following surgery, with the animals standing in a quiet research room. A period of 5 to 10 minutes was allowed for adaptation of the animals to the face mask. The initial recordings and measurements were made with the free end of the Y-connector capped, which allowed respiration only across the glottis and upper airway, and subsequently with the free end of the Y-connector opened, after a 5- to 10-minute period of adaptation to the open Y-connector.

Review of the tracings allowed measurement of respiratory rate, expiratory pressure measured as the peak positive-pressure deflection obtained during the expiratory phase of respiration, inspiratory pressure measured as the peak negative-pressure deflection obtained during the inspiratory phase of respiration, expiratory time, inspiratory time, and the inspiratory-time-to-expiratory-time ratio (I:E). An average value for the variables was obtained from four consecutive respiratory cycles. The respiratory rate was obtained from the flow signal by measuring the total breath duration and dividing it into 60 seconds to obtain a value with a unit of measure of breaths/min. For purposes of this study, expiratory pressure was considered to be equivalent to subglottic pressure.

Twenty-four hours following the baseline measurements, oleic acid (0.08 mL/kg) was admini-

stered via the external jugular vein, and the animals were observed for 24 hours. The previous recordings and measurements were then repeated, first with the Kistner buttons closed to atmosphere and then with them open. Arterial blood gas samples were also obtained during the same conditions with the Kistner buttons open to atmosphere for 20 minutes prior to sampling. The arterial blood samples were placed in ice and analyzed within 5 minutes by a trained laboratory technician using a blood gas analyzer. Arterial blood gases were not measured at baseline in order to minimize trauma to the carotid artery transposition. At the completion of the experimental protocol, a veterinarian (RB) performed necropsy on all animals following euthanasia, with attention to the pulmonary system.

Statistics

Multiple analysis of variance with two repeated measures (before and after oleic acid, and with and without the Kistner buttons open) was employed, using a commercial statistical package.[13] A Bonferroni correction was applied to account for the effect of multiple comparisons. All results are expressed as the mean (standard deviation), and $p \leq 0.05$ was considered significant.

Results

For the animals at baseline (Table 1) with the Kistner buttons closed, the mean value for subglottic pressures was 3.5 cm H_2O [0.34 kPa], which was significantly larger than the value of 1.4 cm H_2O [0.14 kPa] obtained with the Kistner buttons open to atmosphere. No significant differences existed in respiratory rates, inspiratory times, expiratory times, or I:E; however, inspiratory pressures significantly decreased when the Kistner buttons were opened to atmosphere. The results obtained with acute lung injury are shown in the lower part of Table 1. Expiratory pressures, inspiratory pressures, and expiratory times all significantly de-

Table 1. Airway Pressures and Respiratory-Phase Times in Six Healthy Sheep, at Baseline and during Acute Lung Injury

Kistner Button	Respiratory Rate, breaths/min	P_E,* cm H_2O [kPa]	P_I, cm H_2O [kPa]	t_e, ms	t_i, ms	I:E
At Baseline						
Closed to atmosphere	58 (32)†	3.5 (1.4) [0.34 (0.14)]	–4.6 (1.2) [–0.45 (0.12)]	710 (466)	710 (381)	1.09 (0.45)
Open to atmosphere	57 (31)	1.4 (0.5) [0.14 (0.05)]	–1.3 (0.6) [–0.13 (0.06)]	546 (241)	721 (289)	1.47 (0.80)
p value	0.9057	< 0.0001‡	< 0.0001‡	0.1365	0.9055	0.0513
During Acute Lung Injury						
Closed to atmosphere	72 (25)†	12.3 (5.6) [1.21 (0.55)]	–7.3 (5.7) [0.72 (0.56)]	623 (279)	305 (55)	0.59 (0.26)
Open to atmosphere	81 (21)	2.8 (3.7) [0.27 (0.36)]	–3.1 (2.9) [0.30 (0.28)]	398 (214)	406 (103)	1.18 (0.42)
p value	0.1698	< 0.0001‡	0.0028§	0.0031§	0.0002‡	< 0.0001‡

* P_E = expiratory pressure; P_I = inspiratory pressure; t_e = expiratory time; t_i = inspiratory time; I:E = ratio of inspiratory time to expiratory time.

† Values are mean (SD).

‡ p = 0.001 for Bonferroni correction.

§ p = 0.05 for Bonferroni correction.

creased, and inspiratory times and I:E both significantly increased with the Kistner buttons open to atmosphere.

Figure 2 demonstrates the airway pressure tracings and airflow patterns for one of the experimental animals obtained during ALI during breathing with the Kistner button closed to atmosphere (left side of tracings) and then breathing with the Kistner button open to atmosphere (right side of tracings). This representative tracing illustrates the decrease in expiratory or subglottic pressure (lower panel) and the decrease in airflow measured at the mouth (upper panel) that occurred when the Kistner buttons were opened during ALI.

Comparison of the baseline results to the data obtained after ALI with the Kistner buttons closed to atmosphere (the upper lines of data from Table 1) showed no significant difference in respiratory rate with ALI ($p = 0.1033$). Expiratory and inspiratory pressures were both significantly larger with ALI compared to baseline ($p < 0.0001$ and 0.0310, respectively), whereas the expiratory times were found to be not significantly different between the groups ($p = 0.4393$). Both the inspiratory times and the I:E were significantly decreased with ALI compared to baseline ($p < 0.0001$ for both). Both at baseline and after ALI, the sheep developed a reverse I:E when the Kistner buttons were opened to atmosphere. The magnitude of change in the I:E was greatest during ALI (Table 1), when it went from a mean of 0.59 to a mean of 1.18.

Table 2. Arterial Blood Gas Values during Acute Lung Injury (n = 4)

Kistner Button	pH	P_{aO_2},* torr [kPa]	P_{aCO_2}, torr [kPa]
Closed to atmosphere	7.53 (0.02)†	64.3 (4.3) [8.58 (0.57)]	32.5 (6.6) [4.33(0.880)]
Open to atmosphere	7.51 (0.03)	53.5 (8.4) [7.13 (1.12)]	36.3 (6.6) [4.84 (0.88)]
p Value	0.3229	0.0401	0.3471

* P_{aO_2} = partial pressure of arterial oxygen. P_{aCO_2} = partial pressure of arterial carbon dioxide.
† Values are mean (SD).

Arterial blood gas data were obtained in four animals. Two animals were not analyzed due to difficulty with obtaining the samples from the carotid artery transposition in one and the clotting of a sample in the other. The average P_{aO_2} with the Kistner buttons closed was 64.3 (4.3) torr [8.58 (0.57) kPa], which was significantly larger than the value of 53.5 (8.4) torr [7.13 (1.12) kPa] obtained with the Kistner buttons open to atmosphere ($p = 0.0401$). No significant difference existed in the values for pH or P_{aCO_2} (Table 2).

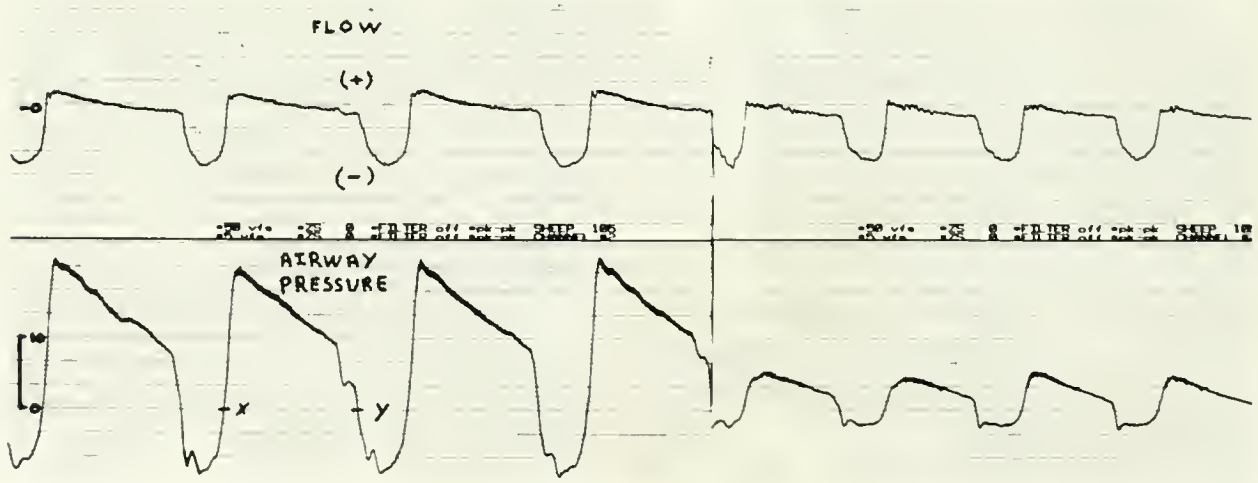

Fig. 2. Airway pressure tracings (bottom) and airflow tracings (top) for one of the experimental animals during acute lung injury, with the Kistner button closed to atmosphere (left side) and then opened to atmosphere (right side). The scale for airway pressure is cm H_2O. x is the start of expiration, and y is the end of expiration. The symbol + indicates the direction of expiratory airflow, and the symbol - indicates the direction of inspiratory airflow.

Review of the necropsy tissue demonstrated evidence of acute lung injury in all animals. Gross examination showed the lungs to be congested, with weeping of edema fluid from cut surfaces. Microscopic examination revealed evidence of diffuse alveolar damage, with interstitial and alveolar edema and pulmonary hemorrhage.

Discussion

Smith et al recently investigated the effect of upper-airway bypass during ALI in a small-animal model.[14] These authors received criticism in an accompanying editorial[15] due to the confounding effects of tracheal intubation, which served as the bypass conduit for the upper airway. The deterioration in gas exchange demonstrated with tracheal intubation in Smith et al's animal study could have been caused by ventilation-perfusion mismatch produced by reflexive distal-airways bronchoconstriction.[15] Our animal model eliminated the influence of tracheal intubation on airways tone, which is thought to be mediated via the vagus nerve.[16-18]

The glottis appears to play an important role in maintaining airway pressures and functional residual capacity (FRC).[19,20] This function is overcome by the placement of an endotracheal tube, as is most easily demonstrated in infants with the removal of the grunting mechanisms that have been shown to be important in maintaining thoracic gas volumes.[6,8] The role of the upper airway in developing subglottic pressure and regulating expiratory airflow is also important for the process of phonation.[20] In addition to vocal-cord movements, the movements of the respiratory bellows provide the subglottic pressure and glottic airflow required for sound generation.[12] As previously stated, subglottic pressure normally approximates alveolar pressure, due to the small pressure drops that occur from the alveoli to the subglottic region.[12] Therefore, subglottic pressure can be approximated as the sum of pleural pressure and the static elastic recoil pressure of the lung.[12] Direct and indirect measurements of subglottic pressure have been performed, using tracheal needle puncture, catheters passed via the glottis, and esophageal balloons.[12,21-24]

The increases in subglottic pressure that occurred in our sheep with ALI are not surprising, due to the decreased lung compliance producing increased static elastic recoil of these injured lungs. Additionally, with ALI, increased respiratory efforts would be expected, which could increase the pleural pressure. Analysis of our airway-pressure tracings (Fig. 2) showed that the elevated subglottic pressures achieved during ALI dramatically decreased with opening of the Kistner buttons. This supports a direct role of the upper airway in maintaining subglottic pressure levels during ALI.

The peak values for subglottic pressure occurred in the earlier phases of expiration. This finding is consistent with the reported normal functioning of the glottis, which usually has a smaller orifice during early expiration than during late or end-expiration.[25-28] The data from Table 1 show that expiratory times significantly decreased and the I:E significantly increased only with ALI, indicating that the upper airway plays a role as an expiratory brake primarily during ALI. At baseline, the mean decrease in expiratory pressure (P_E) with the opening of the Kistner button was only 2.1 cm H_2O [0.21 kPa], whereas the mean decrease with ALI was 9.5 cm H_2O [0.93 kPa]. The relative differences in subglottic pressures occurring with the opening of the Kistner buttons also suggest that the expiratory braking effects of the upper airway are most important during ALI. Because the P_{aO_2} was not measured at baseline, the clinical significance of the small difference in P_E found at baseline with opening of the Kistner buttons is unknown.

Several important limitations of our investigation need to be addressed. We did not measure pleural pressure, elastic recoil pressure, or lung volumes. It is probable that changes in these factors contributed to the generation of higher subglottic pressures during ALI. The use of a Y-connector at the Kistner button site also placed a limitation on our results in that this technique allows measurement of lateral pressures, and not true subglottic pressure, at the point where flow is vented. This would be expected to give lower values for subglottic pressure than actually exist. However, the decrease in subglottic pressure with ALI paralleled the changes seen in expiratory times and arterial oxygen tension, supporting a true decrease in its value. Lung-volume measurements would have

been important in completely assessing these results because decreases in lung volume due to decreases in subglottic pressure with the Kistner buttons open may have accounted for the lower P_{aO_2} values obtained.

Other important limitations of our investigation were that we did not directly assess the relationship between the respiratory muscles and the upper airway during expiratory braking, nor did we examine the effects of time on oxygenation. Remmers and Bartlett, using a small-animal model, demonstrated that opening of a bypass tract to the upper airway was associated with a slowing of the return of transdiaphragmatic pressures from their most negative values to zero, along with an augmentation and prolongation of the diaphragmatic EMG recordings.[29] These results and the work of others[30-32] indicate that respiratory muscles may elicit a compensatory response during bypass of the upper airway to offset the loss of expiratory braking. Our data suggest that compensatory tracking mechanisms such as these may be inadequate during ALI, as evidenced by the decrease in expiratory times and arterial oxygen tensions obtained with opening of the Kistner buttons to atmosphere. The decrease in P_{aO_2} seen during ALI with the Kistner buttons open may also have represented an effect of worsening lung function in our animals, and not an effect of upper-airway bypass. Although the actual time interval between arterial blood gas samples was less than 1 hour, this may have been enough time for progression of the ALI to account for these changes in oxygenation.

The reverse I:E that occurred during ALI with opening of the Kistner button was an unexpected finding in this study. It appeared to be associated with changes in oxygenation, as reflected by the decreased P_{aO_2} values obtained after the opening of the Kistner buttons to atmosphere. This finding may have important implications for critically ill patients with acute lung injury who undergo bypass of the upper airway, as seen during weaning from mechanical ventilation. The reverse I:E produced by upper-airway bypass produces less of the positive pressure in the airways that normally assists in the maintenance of lung volumes, recruitment of alveoli, and maintenance of gas exchange.[33] The prolonged expiratory time produced by the upper airway can be compared to inverse-ratio ventilation (IRV), which extends the time during which positive pressure is applied (during inspiration) and, in theory, maintains or improves gas exchange due to more homogenous lung recruitment.[33] In addition, our findings lend physiologic support to the clinical studies demonstrating improved oxygenation in adults with IRV compared to conventional ventilation.[34,35] The glottis appears to produce effects similar to those in IRV except that positive airway pressures are produced during expiration and not during inspiration.

Conclusions

In adults, the role of the glottis appears to be an active one, allowing for braking or buttressing of the expiratory airflow generated by respiratory muscle activity and the static elastic recoil pressure of the lung.[27,36] Our results suggest that (1) partial bypass of the upper airway may be associated with a decrease in the development of subglottic pressures, and (2) respiratory timing and pulmonary gas exchange are influenced by the upper airway during ALI. The full significance of upper-airway function during ALI has not been adequately investigated. Future studies should examine the changes that occur in diaphragmatic function, intrapleural-pressure generation, elastic recoil pressure, and the development of upper-airway edema, to assess their roles in lung function during ALI.

PRODUCT SOURCES

Medications:
Telazol, tiletamine hydrochloride, and zolazepam hydrochloride, AH Robins Co, Richmond VA

Xylocaine, lidocaine hydrochloride, Astra Pharmaceutical Products Inc, Westborough MA

Pulse Oximeter:
Ohmeda Model 3710, Ohmeda, Madison WI

Tracheal Button:
Kistner Button, Pilling Corp, Fort Washington PA

Pneumotachograph:
Fleisch Model 0, dead space 4.7 mL, Metabo Co, Switzerland

Pressure Transducers:
Statham, Hato Rey, Puerto Rico

Dual-Bridge Amplifier:
Fogg Model 262, Fogg System Co Inc, Denver CO

Waveform Recorder:
Model DASH VI. Astro-Med Inc. West Warwick RI

Blood Gas Analyzer:
BGE Model 1400, Instrumentation Laboratory, Lexington MA

Statistical Software Package:
BMDP Statistical Software Manual. University of California Press, Berkeley CA

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REFERENCES

1. Weisman IM, Rinaldo JE, Rodgers RM. Positive end-expiratory pressure in adult respiratory failure. N Engl J Med 1982;307:1381-1384.
2. Feeley TW, Saumarez R, Klick JM, McNabb TG, Skillman JJ. Positive end-expiratory pressure in weaning patients from controlled ventilation: a prospective randomized trial. Lancet 1975;2:725-729.
3. Annest SJ, Gottlieb M, Paloski WH, Stratton H, Newell JC, Dutton R, et al. Detrimental effects of removing end-expiratory pressure prior to endotracheal extubation. Ann Surg 1980;191:539-545.
4. Venus B, Copiozo GB, Jacobs HK. Continuous positive airway pressure: the use of low levels in adult patients with artificial airways. Arch Surg 1980;115:824-828.
5. Quan SF, Falltrick RT, Schlobohm RM. Extubation from ambient or expiratory positive airway pressure in adults. Anesthesiology 1981;55:53-56.
6. Fox WW, Berman LS, Dinwiddie R, Shaffer TH. Tracheal extubation of the neonate at 2 to 3 cm H_2O continuous positive airway pressure. Pediatrics 1977;59: 257-261.
7. Berman LS, Fox WW, Raphaely RC, Downs JJ. Optimal levels of CPAP for tracheal intubation of newborn infants. J Pediatr 1976;89:109-112.
8. Harrison VC, Heese HdeV, Klein M. The significance of grunting in hyaline membrane disease. Pediatrics 1968;41:549-559.
9. Gal TJ, Suratt PM. Resistance to breathing in healthy subjects following endotracheal intubation under topical anesthesia. Anesth Analg 1980;59:270-274.
10. Dohi S, Gold MI. Pulmonary mechanics during general anaesthesia: the influence of mechanical irritation on the airway. Br J Anaesth 1979;51:205-214.
11. Gal TJ. Pulmonary mechanics in normal subjects following endotracheal intubation. Anesthesiology 1980; 52:27-35.
12. Bouhuys A, Proctor DF, Mead J. Kinetic aspects of singing. J Appl Physiol 1966;21:483-496.
13. Ryan-Wenger NM. Guidelines for critique of a research report. Heart Lung 1992;21:394-401.
14. Smith RA, Venus B, Johnson MT, Carter C. Influence of glottic mechanism on pulmonary function after acute lung injury. Chest 1990;98:206-208.
15. Gal TJ. Is glottic function the key to improved gas exchange? (editorial). Chest 1990;98:9-10.
16. Guz AA. Respiratory sensation in man. Br Med Bull 1977;33:175-177.
17. Nadel JA, Cabezas GA, Austin JH. In vivo roentgenographic examination of parasympathetic innervation of the small airways. Invest Radiol 1971;6:9-17.
18. Woolcock AJ, Macklem PT, Hogg JC, Wilson NJ, Nadel JA, Frank NR, et al. Effect of vagal stimulation on central and peripheral airways in dogs. J Appl Physiol 1969;26:806-813.
19. Campbell AH, Imberger H, Jones BM. Increased upper airway resistance in patients with airway narrowing. Br J Dis Chest 1976;70:58-65.
20. Strenger F. Methods for direct and indirect measurement of the subglottic air pressure in phonation. Studia Linguistica 1960;14:98-112.
21. Isshiki N. Regulatory mechanism of voice intensity variation. J Speech Hearing Res 1964;7:17-29.
22. Van den Berg J. Direct and indirect determination of the mean subglottic air pressure. Folia Phoniat 1956;8:1-24.
23. Draper MH, Ladefoged P, Whitterridge D. Respiratory muscles in speech. J Speech Hearing Res 1959;2:16-27.
24. Draper MH, Ladefoged P, Whitteridge D. Expiratory pressure and air flow during speech. Br Med J 1960; 1:1837-1843.
25. Dixon M, Swerda-Przestaszewska M, Widdicombe JG, Wise JCM. Studies on laryngeal calibre during stimulation of peripheral and central chemoreceptors, pneumothorax and increased respiratory loads. J Physiol (Lond) 1974;239:347-363.
26. Gautier H, Remmers JE, Bartlett D Jr. Control of the duration of expiration. Respir Physiol 1973;18:205-221.
27. Spann RW, Hyatt RE. Factors affecting upper airway resistance in conscious man. J Appl Physiol 1971;31:708-712.
28. Baier H, Wanner A, Zarzecki S, Sackner MA. Relationships among glottis opening, respiratory flow, and upper airway resistance in humans. J Appl Physiol 1977;43:603-611.
29. Remmers JE, Bartlett D. Reflex control of expiratory airflow and duration. J Appl Physiol 1977;42:80-87.
30. England SJ, Bartlett D Jr. Changes in respiratory movements of the human vocal cords during hyperpnea. J Appl Physiol 1982;52:780-785.
31. Bishop B, Bacofen H. Comparison of neural control of diaphragm and abdominal muscle activities in the cat. J Appl Physiol 1972;32:798-805.
32. Campbell EJM, Green JH. The behavior of the abdominal muscles and the intra-abdominal pressure during quiet breathing and increased pulmonary ventilation: a study in man. J Physiol (Lond) 1955;127:423-426.

33. Marcy TW, Marini JJ. Inverse ratio ventilation in ARDS: rationale and implementation. Chest 1991;100: 494-504.
34. Tharratt R, Allen R, Albertson T. Pressure controlled inverse ratio ventilation in severe adult respiratory failure. Chest 1988;94:755-762.
35. Gurevitch M, Van Dyke J, Young E, Jackson K. Improved oxygenation and lower peak airway pressure in severe adult respiratory distress syndrome: treatment with inverse ratio ventilation. Chest 1986;89:211-213.
36. England SJ, Bartlett D Jr, Daubenspeck JA. Influences of human vocal cord movement on airflow and resistance during eupnea. J Appl Physiol 1982;52:773-779.

Pulmonary Vascular Smooth-Muscle Regulation: The Role of Inhaled Nitric Oxide Gas

Christopher C Miller BA RRT and John WR Miller BS

I. Introduction
II. Vascular Smooth-Muscle Contraction and Relaxation
III. Cyclic Guanosine 3', 5' Monophosphate and Endothelium-Derived Relaxing Factor
IV. Nitric Oxide Gas: A Selective Pulmonary Vasodilator
V. Properties of Nitric Oxide
VI. Future Directions

Introduction

It is hard to imagine a toxic air pollutant juxtaposed with important medical gases like oxygen and nitrous oxide. Yet, the possible use of inhaled nitric oxide (NO) as a therapeutic gas may well be one of the more important findings in pulmonary medicine. If this proves to be the case, a revolution in medical gas therapy may result. Respiratory care practitioners (RCP), already knowledgeable of and adept at gas delivery, would be expected to play an important role should this new therapy come to fruition.

Even if NO gas therapy does not prove to be a panacea for pulmonary vascular hypertensive disease, it is still likely that NO gas will be used clinically in selected disease states such as adult respiratory distress syndrome and persistent pulmonary hypertension of the newborn.[1] In order to understand the importance of inhaled NO gas as a selective pulmonary vasodilator, a review of important postulates set forth by previous research is necessary. This paper examines the accepted model of vascular smooth-muscle contraction, explains the role of guanosine 3', 5' monophosphate (cyclic GMP, or cGMP) and endothelium-derived relaxing factor (EDRF) in vascular smooth-muscle relaxation, and presents challenges that must be faced prior to therapeutic use of NO gas. A better understanding of these issues should clarify why the use of NO as an inhaled medical gas is currently a topic of interest in pulmonary medicine.

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Vascular Smooth-Muscle Contraction and Relaxation

The mechanisms involved in regulation and contraction of smooth muscle are poorly understood and controversial at best. In general, vascular smooth-muscle tone can be altered by a complex interplay among nervous, hormonal, chemical, electrical, and physical factors. Conclusions from extensive literature reviews from the late 1970s still form the basis of today's model of smooth-muscle contraction.[2,3] This model suggests that vascular smooth-muscle contraction is ultimately dependent upon the presence of intracellular (cytoplasmic) calcium ion concentration (Ca^{++}) and the protein calmodulin.[4,5] Specifically, all factors affecting contractility and relaxation act either directly or indirectly to increase or decrease cytoplasmic Ca^{++}.[6]

In review, let us compare what is known about smooth muscle with its counterpart skeletal, or striated, muscle. Striations in skeletal muscle are visible under a normal light microscope, and their visibility is due to alternating bands of light and dark areas perpendicular to the longitudinal myofibrils (Z and M lines; H, I, and A bands). Myofibrils are the threadlike contractile units of muscle fiber. It is well-established that myofibrils in striated muscles are made up of thick filaments composed of the protein myosin and thin filaments, which are a composite of three proteins: actin, tropomyosin, and troponin. The overlapping of the thick and thin filaments is responsible for the striated appearance (Fig. 1).

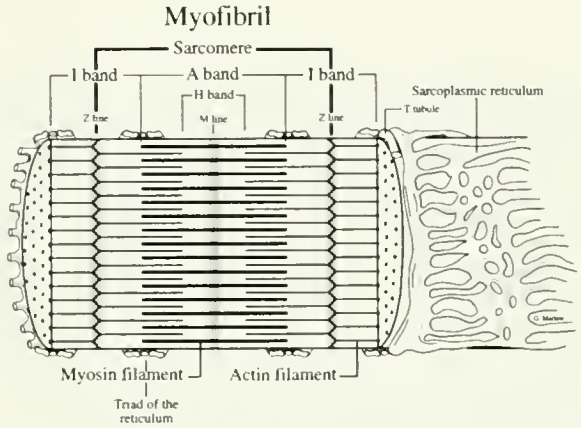

Fig. 1. Diagrammatic representation of myofibrils of skeletal muscle fiber. The A band corresponds to thick myosin filaments overlapped in the darker regions with thin actin filaments; the lighter H band contains only thick myosin filaments. I bands represent nonoverlapping areas of thin actin filaments attached to Z lines. The area between two adjacent Z lines is called a sarcomere. The sarcoplasmic reticulum is a tubular network that runs parallel to the myofibrils and encases them.

In smooth muscle, no cross-striations are visible. A possible reason for this difference is that smooth-muscle filaments are not organized in regular parallel arrays as is striated muscle. In striated muscle, the ends of the thin actin filaments are anchored in a perpendicular dark-staining region known as the Z-line (sarcomere). Between two Z-lines is a region known as a sarcomere, which contains the M line and H and A bands. In smooth muscle, actin ends (F-actin) radiate out of dense bundles. These bundles are distributed at periodic intervals throughout the cell, and no detectable Z-lines exist.[7] These dense areas secure the F-actin ends as do Z lines in skeletal muscle. The other end of the actin filament (G-actin) is globular and participates in myosin-binding during muscle contraction.

Another reason for a lack of banding in smooth muscle may be due to the different chemical make-up of the thin actin filament. Unlike striated muscle, smooth-muscle actin filaments contain tropomyosin but not troponin. Troponin participates in the binding of Ca^{++} in skeletal muscle contraction. Troponin is replaced in smooth muscle by other Ca^{++}-binding proteins called calmodulin and leiotonin.[8]

The mechanism of muscle contraction in striated muscles seems to apply also to smooth muscles. A generally accepted theory for the shortening of muscle fiber is based upon the "sliding filament" model.[9,10] Briefly, contraction is the function of the physical interaction between the proteins actin and myosin. Actin proteins form long helical structures that run parallel to each other and are sandwiched between myosin fibrils. The heads of the myosin proteins bend and swivel to attach to the myosin-binding sites on actin (G-actin). The area of this biochemical interaction on the head of the myosin fibril is referred to as the myosin-P (phosphorylatable) light chain.[7] As contraction continues, the myosin-P light-chain heads detach, reattach to the G-actin, and continue to 'slide' along the actin chain in a ratcheting fashion.[11] The bending and swiveling action of myosin heads requires energy.

The energy needed for this important contractile mechanism is supplied by the hydrolysis of adenosine triphosphate (ATP) into adenosine diphosphate (ADP) on the phosphorylated head of the myosin-P light chain.[12] ATP can be catalyzed to break (hydrolyze) a high energy phosphate (P) bond, releasing large amounts of energy. The energy released drives the chemical reaction that causes the bending and swiveling of the myosin-P light-chain head in the process of muscle contraction. The resulting ADP can be rephosphorylated using energy provided from glucose or fatty acids.[13-15] Muscle contraction is activated when a depolarizing stimulus (neural, electrical, humoral, or physical) initiates the release of Ca^{++} from the sarcoplasmic reticulum that surrounds the myofibrils.

Two models have been proposed to explain how Ca^{++} triggers smooth-muscle contraction—the phosphorylation model and the calcium direct-

binding model.[16] Evidence is accumulating that both models may modulate different aspects of the actin-myosin interaction; phosphorylation regulates the velocity of the shortening while Ca^{++} binding regulates the production of sustained force.[17,18]

The phosphorylation model proposes that cytoplasmic Ca^{++} binds with calmodulin in the smooth-muscle cell. The calmodulin-Ca^{+} complex then activates a myosin light-chain kinase. This enzyme catalyzes the phosphorylation of the myosin-P light-chain heads, and muscle contraction proceeds according to the sliding filament theory. According to the Ca^{++}-binding model, contraction is sustained until Ca^{++} is reabsorbed into the sarcoplasmic reticulum.[17,18]

As described previously, the distal ends of the actin (F-actin) filaments are fixed at the center of the dense Z-like end of the sarcomere and radiate outwards like the spines of a sea urchin. The overlapping of the actin fibril sliding along the myosin filament causes a shortening of the muscle fiber length towards the central hub. Irregular locations of these Z-like plates in smooth muscle cause a shortening of the muscle width as well as length. This three-dimensional contraction causes an enfolding or puckering of the smooth-muscle wall.[7] The result is a necessary attribute of vascular smooth muscles, affecting the diameter of the vessel wall (Fig. 2).

The main storage sites for intracellular Ca^{++} are the sarcoplasmic reticulum and the plasma membrane. In addition, Ca^{++} can rapidly enter the smooth-muscle cell through voltage-gated calcium channels and receptor-operated ion channels. However, the sarcoplasmic reticulum normally has a calcium pump that accumulates intracellular calcium when the cell is at rest. The sarcoplasmic reticulum and the plasma membrane generally store enough calcium to allow the smooth-muscle cell to contract for some time, even in the absence of extracellular calcium.[7]

An understanding of smooth-muscle contraction helps us to comprehend its antithesis, smooth-muscle relaxation. It follows that anything that promulgates the absorption of free cytoplasmic Ca^{++} will thus inhibit contraction. Cyclic guanosine 3', 5' monophosphate (cyclic GMP) is one such intracellular substance that reduces cytoplasmic Ca^{++} levels. Endothelium-derived relaxing factor (EDRF) is a substance produced in the endothelial cells that

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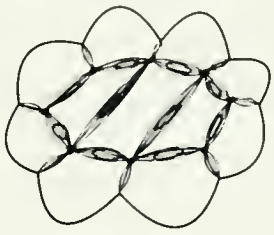

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Fig. 2. Schematic representation showing contraction in a smooth-muscle cell. Actin filaments are anchored in the intersections and slide along the myosin filaments that run parallel along these lines. Upon contraction, the distance between the dense bodies shortens and the cell wall balloons out causing puckering on its surface. This process is responsible for decreasing the diameter of the vessel wall. (Reproduced, with permission, from Reference 7.)

increases the level of cyclic GMP in adjacent smooth-muscle cells. Cyclic GMP and EDRF are perhaps the two most important compounds modulating smooth-muscle tone.

Cyclic Guanosine 3', 5' Monophosphate and Endothelium-Derived Relaxing Factor

In the mid-1970s, it was thought that cyclic guanosine 3', 5' monophosphate (cGMP) was actually involved in smooth-muscle contraction.[19,20] The work of Ignarro and others dispelled this notion.[21-23] By the mid-1980s, it was generally accepted that increases in intracellular cGMP levels preceded and were directly linked to the process of smooth-muscle relaxation. Higher levels of cGMP decrease intracellular Ca^{++} levels by preventing the release of bound Ca^{++} from the sarcoplasmic reticulum in the smooth-muscle cell.[24,25] The exact means by which increased levels of cGMP reduce cytoplasmic Ca^{++} levels still remain vague.[7,26] Further research during the same period proposed and supported the hypothesis that compounds capable of releasing or forming nitric oxide activated guanylate cyclase, which catalyzes the production of cGMP.[27-30] In 1985, Ignarro hypothesized the mechanism of this action, and it can be summarized as follows:[31] Nitrogen oxide-containing vaso-

dilators such as nitroprusside, nitroglycerin, and other related drugs cause smooth-muscle relaxation. These compounds permeate the smooth-muscle-cell wall. Once inside the cell, they generate intracellular intermediates called S-nitrosothiols.[3,4,31-33] These substances rapidly decompose to form NO gas. NO gas then activates the enzyme guanylate cyclase, which produces cGMP. The cytoplasmic level of cGMP rises and prevents the release of Ca^{++} from the sarcoplasmic reticulum. The decreased availability of intracellular Ca^{++} inhibits the formation of the calmodulin-Ca^{++} complex. Phosphorylation of the myosin-P light chain is inhibited, consequently attenuating vascular smooth-muscle contraction.

The walls of arteries are lined with endothelial cells that lie adjacent to smooth-muscle cells.[32] In 1975, Moncada and co-workers discovered these endothelial cells to be necessary for vascular smooth-muscle relaxation.[33] Furchgott and Zawadzki found in 1980 that the cellular depolarizing action of acetylcholine stimulated the release of a powerful smooth-muscle relaxing agent in the endothelial cell. They named this substance endothelium-derived relaxing factor (EDRF).[34] They further concluded that arterial smooth-muscle contraction is dependent upon an intact endothelium to produce EDRF. The endothelium also produces potent vasoconstrictors.[35] Dinh Xuan and co-workers have suggested that vascular muscle tone is dynamic and represents a balance between "vasorelaxing" and "vasoconstricting" influences.[36] A wide range of endothelium-dependent drugs have since been used in the clinical setting as vasodilators.[31] However, only recently has their vasodilating action been linked to EDRF.

Throughout the mid-1980s, reports revealed that NO gas was either a major component of EDRF or NO gas alone was EDRF.[37-39] Bioassay studies have since shown that EDRF and NO are highly diffusible gases with very short half lives in aqueous buffered solutions.[37,40,41] Two research groups simultaneously proposed that EDRF and NO gas were the same.[38,42] Although it is still debated whether these two are identical active species, NO or a NO-producing compound is now recognized as a major vasodilator originating from the endothelium.[38,42-44] The hypothesis that inhaled NO gas is a selective pulmonary vasodilator is based upon this premise.

Nitric Oxide Gas: A Selective Pulmonary Vasodilator

Regional constriction of the pulmonary vascular bed is a normal physiologic response.[45] This protective reflex attempts to preserve the ideal ventilation-to-perfusion ratio ($\dot{V}/\dot{Q}$) between 1.0 and 0.8.[46] $\dot{V}/\dot{Q}$ mismatching is the most common cause of hypoxemia in the critically ill.[47] Modulation of pulmonary vascular tone is a localized phenomenon and is independent of neural and humoral stimuli.[48] On occasion, excessive pulmonary vasoconstriction may inadvertently contribute to a severe $\dot{V}/\dot{Q}$ inequality that may lead to the demise of the patient. Adult respiratory distress syndrome, persistent pulmonary hypertension of the newborn, and pulmonary hypertension secondary to cardiac disease are but a few of these pathologies. An important fraction of the critically ill present with or develop these problems and have high morbidity.

Various drugs have been used to reduce pulmonary vascular resistance. These include but are not limited to arachidonic acid metabolites, organic nitrates, organic nitrites, organic nitroso- compounds, acetylcholine, calcium channel blockers, and magnesium sulfate.[29,31,49-53] The pharmacologic actions of these drugs vary but ultimately result in the decrease of intracellular (cytoplasmic) Ca^{++}, a decrease that causes smooth-muscle relaxation.[6]

The major stumbling block preventing the use of these cytoplasmic calcium inhibitors as pulmonary vasodilators is the inability to achieve specific regional selectivity. Associated with the attenuation of pulmonary vascular tension is a concomitant dilatation of the systemic arterial vasculature—invariably leading to systemic hypotension and various other reported complications.[54-56] Inhaled NO gas may be the ideal solution to this problem because it is inactivated prior to reaching the systemic circulation. If proven to be safe and effective at reducing pulmonary hypertension, inhalation of NO gas may begin a new era in the selective treatment of pulmonary vascular disease.[57]

NO gas, as previously discussed, has been shown to be a potent and rapid, but transient, smooth-muscle relaxant. Recent literature supports the hypothesis that endogenous NO gas may be an important modulator of vascular smooth-muscle control.[58-62] This hypothesis is summarized in the following paragraph and in Figure 3.

Fig. 3. Diagrammatic representation of the proposed biocascade mechanism of endothelium-dependent nitric oxide gas (NO) modulating smooth-muscle tone. NO diffusing into the smooth muscle attenuates muscle contraction, whereas NO entering the blood stream is rapidly inactivated by hemoglobin.

NO gas and EDRF appear to possess identical activity.[34,37-39,63] Nitric oxide synthase[64-67] converts L-arginine[67-70] into NO gas and citrulline[71] in the endothelial cells. NO gas then diffuses into the adjacent smooth muscle where it, directly as NO gas or indirectly as an intermediate S-nitrosothiol,[29,30,50,72,73] activates guanylate cyclase.[74-78] The presence of the enzyme guanylate cyclase increases cGMP, causing smooth-muscle relaxation[51,74-78] by preventing the release of Ca^{++} from the sarcoplasmic reticulum.[24,25] In the intravascular space, NO gas immediately binds with the heme ring of hemoglobin to form nitrosylhemoglobin[79,80] Oxidation occurs forming methemoglobin.[81,82] Methemoglobin is subsequently reduced to nitrite and nitrates, which are excreted in the urine.

The importance of NO gas as a pulmonary vascular muscle relaxant rests on its characteristic of being delivered as a gas directly to the pulmonary vascular system, without affecting systemic vascular tone. Like oxygen, NO gas can be administered via the pulmonary airways. The proximity of the alveoli to the pulmonary vascular smooth muscle suggests that NO gas acts by diffusing across the alveolar membrane into the smooth muscle, attenuating pulmonary vascular smooth-muscle tone[32] by the same chemical mechanism as discussed previously. The primary advantage of delivering NO gas directly to the pulmonary vascular system with ventilation is that it will not reach the systemic circulation (Fig. 4).

Specifically, NO gas, which passes beyond the pulmonary smooth muscle into the luminal side of the vasculature, is instantly inactivated by hemoglobin.[83] It has been shown that NO gas diffuses readily across the alveolocapillary barrier and has a very high affinity for hemoglobin.[81-83] In fact, NO gas binds 280 times faster than carbon monoxide.[84] For this reason, as evidenced by recent research, NO gas might also be a better alternative gas for pulmonary diffusion studies.[83] Once NO gas penetrates beyond the smooth-muscle cell into the circulating blood, it immediately reacts with hemoglobin forming methemoglobin. It is known that

Fig. 4. Diagrammatic illustration showing how nitric oxide gas (NO) delivered from the pulmonary airways might promote muscle relaxation and avoid the side effects of systemic vasodilatation.

smokers have increased methemoglobin levels as compared to nonsmokers (a fact that can now be explained by this NO gas-hemoglobin reaction[85]). Inhaled cigarette smoke contains between 400 and 1,000 ppm of NO gas and is reduced by hemoglobin into methemoglobin.[86]

In summary, inhalation of NO gas would achieve pulmonary dilatation by diffusing directly from the alveoli into the vascular smooth-muscle cells causing relaxation. The problem of concomitant dilatation of the systemic vasculature seen with intravenously administered vasodilators is avoided because NO gas permeating beyond the pulmonary smooth muscle is instantaneously inactivated by hemoglobin. Correct titration of NO gas appears to be of paramount importance. Too little may not achieve the desired result and too much might limit oxygen content due to excessive methemoglobin levels. It also remains to be seen whether NO gas dilates bronchial smooth muscle, especially in patients with small airways disease.

Because NO gas is delivered from the alveolar side, it should selectively increase perfusion only to ventilated areas of the lung—normalizing the $\dot{V}/\dot{Q}$ inequality. This in fact has been shown to occur in patients with adult respiratory distress syndrome.[57,87-89] The result would mean a decreased fractional inspired oxygen concentration (F_{IO_2}) requirement for many patients and a consequent avoidance of oxygen toxicity. If it is shown that inhaled NO gas relieves pulmonary vascular resistance only in ventilated areas of the lung, the prognosis for many intensive care patients suffering from hypoxemia or increased pulmonary hypertension may dramatically improve.[87] Morbidity, secondary to these problems, could also decrease significantly.

NO gas as an inhaled smooth-muscle relaxant has been a topic of major interest at the American Thoracic Society and American Lung Association International Conference, other professional meetings, and in other journals during the last two years (1991-1992).[87-94] Notwithstanding the interest and research supporting the use of inhaled NO gas as a selective pulmonary vasodilator, some important unknowns remain with respect to its application in the human model.

Properties of Nitric Oxide

Despite the popularity of this novel therapeutic approach for treating pulmonary vascular hypertension, a dark side exists, as with most drugs. As one British-trained anesthetist recently remarked (David Rhydderch MD, Anesthesia Dept, King Faisal Specialist Hospital & Research Center, Riyadh, Saudi Arabia, 1992), "It's difficult to adjust to the idea that a gas which we thought might be lethal could now become a lifesaving treatment." A review of the literature provides a reasonable explanation for this guarded acceptance.

In 1966, at the University of Bristol, contamination of a nitrous oxide cylinder with NO gas caused the tragic death of one and the near fatality of another patient.[96] The victims presented with gross hypoxemia, secondary to high levels of methemoglobin and pulmonary edema. It was deduced that the contaminant gas, NO, was delivered along with nitrous oxide anesthesia. Pulmonary edema was apparently caused by the toxic effects of the oxides of NO gas. Excessive levels of methemoglobin in the circulating blood were thought to be the result of NO oxidation by hemoglobin. Following the Bristol report, the purification, storage, and procedures for handling nitrous oxide were scrupulously reviewed, and anesthetists were alerted to the signs and symptoms of exposure to NO gas and its oxides.[97,98] The use of NO as a therapeutic gas will likely be viewed with strong hesitancy. For this reason alone it is important that all clinicians become familiar with the properties, measurement techniques, and toxicology of this gas prior to its use in the clinical sphere.

NO is a colorless, highly diffusible gas with a density about the same as that of air (1.04). In the presence of oxygen, NO gas combines with molecular oxygen to form a red-brown gas called nitrogen dioxide (NO_2). When exposed to NO, NO_2 reacts readily to produce dinitrogen trioxide, which is soluble in water, forming either nitrous or nitric acid.[88] NO_2 is a highly reactive gas and is a toxic irritant to the human lung. In concentrations of 2-5 ppm for a 4-hour period, NO_2 causes mild inflammation in the human lung.[94,99] In the animal model, exposure to high levels of mixed oxides of nitrogen (> 518 ppm) causes considerable immediate damage to the alveolar epithelium. This is followed by a 6-hour latent period culminating in the development of pulmonary edema.[100] A recent review of the risk of human exposure to NO_2 in the form of environmental pollutants from combustion engines warns of toxic effects through increased susceptibility to infection and increased airway deficits.[101] Some of these findings are consistent with the clinical symptoms and postmortem examination of the 1966 Bristol death. Fortunately, NO at low concentrations reacts very slowly with oxygen to form these toxic products. The justification for using NO as a therapeutic gas is based on this rationale.

The reaction of NO with oxygen to form poisonous compounds is dependent on the concentration of oxygen and the square of the concentration of NO.[88] Practically speaking, this means that a 50% conversion of NO to NO_2 at 10 ppm in room air (20°C) takes 7 hours. At 100 ppm it would take 40 minutes. This may explain why no adverse effects have been observed in recent short-term exposure of laboratory animals to concentrations as high as 100 ppm NO.[57,102] In the pulmonary hypertensive human model, similarly encouraging findings have been reported while breathing an air/NO (40 ppm) mixture.[89] The United States Occupational Safety and Health Administration has established the time-weighted-average NO exposure value at 25 ppm.[103] Despite these encouraging preliminary findings, a number of important toxicologic studies remain to be done.

Future Directions

The literature presents a strong argument for the use of inhaled NO gas as a specific pulmonary vasodilator for the purpose of reducing $\dot{V}/\dot{Q}$ inequalities. For the RCP, the most tangible result of using inhaled NO will be its effect on decreasing requirements for ventilated ICU patients. However, it remains to be seen whether NO gas can be delivered safely and effectively to the critically ill. Much research needs to be done, and many questions need to be answered in the immediate future. Specifically, the toxicologic effects of long-term exposure to NO gas must be determined.

Measuring NO gas and its gaseous oxides presents a formidable challenge. It was discovered in the mid-1960s that ozone could be used to detect the presence of NO.[104,105] The reaction of NO with

ozone releases energy in the form of light. This principle is known as chemiluminescence. Measurement of NO and its other oxides is based on this principle. Each oxide of nitrogen reacts with ozone, and a specific measurable wavelength of light is emitted. An increase in the awareness of our environment has helped in the development of various analyzers. Because NO and other nitrogen oxides are common air pollutants, it is not surprising that these measurement techniques exist. However, the application of chemiluminescence in the clinical environment has yet to be addressed, and as late as 1982 was deemed unnecessary.[106] Clearly methods of measurement and analysis of these gas mixtures remain in their infancy.[107,108]

Mixing and delivering various fractional inspired NO gas and oxygen concentrations while avoiding the production of undesired poisonous gases is still to be worked out. Inspired gas mixtures must then be accurately measured to determine the level of oxidant gases for each level. Presently, there are no studies measuring chemical reaction rates of oxides of nitrogen at 37°C. Once the solutions to these challenges are established, a method of delivery to the ventilated patient must be sought. Exhaled patient-gas scrubbers must still be constructed to protect the health-care worker. Predictably, a vast number of studies must be conducted before inhaled NO is accepted as the drug of choice for the treatment of pulmonary hypertension and for the improvement of oxygenation in the critically ill.

Medical gas therapy is a familiar realm for the RCP. Many questions in the field of NO gas therapy could well be addressed and solved by RCPs. The door is just opening on the possibility that NO gas may also be useful in bronchodilator therapy.[95] Surely RCPs will be involved with NO gas therapy should this, too, become a therapeutic modality. A unique opportunity exists for RCPs to rise to the challenge of the occasion—to be on the leading edge of a new medical gas therapy. The intention of this article is to establish an updated understanding of NO gas therapy and to stimulate an interest in an exciting new area of therapy. In conclusion, as Robert Kacmarek recently said, quoting Horace in 23 BC, perhaps this is the opportune moment for RCPs to "Carpe diem!"[109]

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REFERENCES

1. Nitric oxide in the clinical arena (editorial). Lancet 1991;338:1560-1562.
2. Casteels R, Godfrained T, Rvegg JC. Excitation-contraction coupling in smooth muscle. Amsterdam: Elsevier/North Holland, 1979.
3. Dagby RM, Corey-Kreyling MD. Structural aspects of the contractile machinery of smooth muscle. "Is the organization of contractible elements compatible with a sliding filament mechanism?" In: Stephens NL, ed. Smooth muscle contraction. New York: Marcel Dekker, 1984:47-74.
4. Peach MJ, Singer HA, Izzo NJ, Leob AL. Role of calcium in endothelium-dependent relaxation of arterial smooth muscle. Am J Cardiol 1987;59:35A-43A.
5. Weinheimer G, Osswald H. Inhibition of endothelium-dependent smooth muscle relaxation by calmodulin antagonists. Arch Pharmacol 1986;332:391-397.
6. Cutfield GR. The systemic and pulmonary circulations. In: Tinker J, Zapol W, eds. Care of the critically ill patient, 2nd ed. London: Springer-Verlag, 1992:24-38.
7. Gordon AM. Contraction in smooth muscle and non-muscle cells. In: Patton HD, Fuchs AF, Hille B, Scher AM, Steiner R, eds. Textbook of physiology: excitable cells and neurophysiology. London: WB Saunders, 1989:214-229.
8. Nonomura Y, Ebashi S. Calcium regulatory mechanism in vertebrate smooth muscle. Biomed Res 1980;1:1-14.
9. Cook R. The mechanism of muscle contraction. CRC Crit Rev Biochem 1986;21:53-118.
10. Goldman YE, Brenner B. Special topic: molecular mechanism of muscle contraction. Annu Rev Physiol 1987;49:629-636.
11. Gabella G. Structural apparatus for force transmission in smooth muscles. Physiol Rev 1984;64:455-477.
12. Miller-Hance WC, Kamm KE, Stull JJ. Role of calcium and myosin phosphorylation in arterial smooth muscle contraction. In: Aoki K, Frohlich ED, eds. Calcium in essential hypertension. London: Academic Press, 1989: 133-168.
13. Alberts B, Bray D, Lewis J, Raff M, Roberts K, Watson JD, eds. Molecular biology of the cell, 2nd ed. New York: Garland, 1989:64-65.
14. Hess B, Markus M. Order and chaos in biochemistry. Trends Biochem Sci 1987;12:45-48.
15. McGilvery RW. Biochemistry: a functional approach, 3rd ed. Philadelphia: WB Saunders, 1983.
16. Kamm KE, Stull JT. The function of myosin and myosin light chain kinase: phosphorylation in smooth muscle. Annu Rev Pharmacol Toxicol 1985;25:593-620.

17. Cassidy P, Hoar PE, Kerrick WGL. Irreversible thiophosphorylation and activation of tension in functionally skinned rabbit ileum strips by [35S] ATPS. J Biol Chem 1979;254:11148-11153.
18. Murphy RA, Aksoy MO, Dillon PF, Gerthoffer WT, Kamm KE. The role of myosin light chain phosphorylation in regulation of the cross-bridge cycle. Fed Proc 1983;42:51-56.
19. Andersson R, Nilsson K, Wikberg J, Johansson S, Mohme-Lundholm E, Lundholm L. Cyclic nucleotides and the contraction of smooth muscle. Adv Cyclic Nucl Res 1975;5:491-518.
20. Diamond J. Role of cyclic nucleotides in control of smooth muscle contraction. Adv Cyclic Nucl Res 1978; 9:327-340.
21. Bohme E, Graf H, Schultz G. Effects of sodium nitroprusside and other smooth muscle relaxants on cyclic GMP formation in smooth muscle and platelets. Adv Cyclic Nucl Res 1978;9:131-143.
22. Diamond J. Lack of correlation between cyclic GMP elevation and relaxation of nonvascular smooth muscle by nitroglycerin, nitroprusside, hydroxylamine and sodium azide. J Pharmacol Exp Ther 1983;225:422-426.
23. Gruetter CA, Barry BK, McNamara DB, Gruetler DY, Kadowitz PJ, Ignarro LJ. Relaxation of bovine coronary artery and activation of coronary arterial guanylate cyclase by nitric oxide, nitroprusside and carinogenic nitrosoamine. J Cyclic Nucl Res 1979;5:211-224.
24. Collins P, Griffith TM, Henderson AH, Lewis MJ. Endothelium-derived relaxing factor alters calcium fluxes in rabbit aorta: a cyclic guanosine monophosphate-mediated effect. J Physiol 1986;381:427-437.
25. Rapoport RM. Cyclic guanosine monophosphate inhibition of contraction may be mediated through inhibition of phosphatidyl-inositol hydrolysis in rat aorta. Circ Res 1986;58:407-410.
26. Bulbring E, Brading AF, Jones AW, Tomita T. Smooth muscle: an assessment of current knowledge. Austin: University of Texas Press, 1981.
27. Gruetter CA, Barry BK, McNamara DB, Kadowitz PJ, Ignarro LJ. Coronary arterial relaxation and guanylate cyclase activation by cigarette smoke, N'-nitrosonornicotine and nitric oxide. J Pharmacol Exp Ther 1980;214:9-15.
28. Gruetter CA, Gruetter DY, Lyon JE, Kadowitz PJ, Ignarro LJ. Relationship between cyclic GMP formation and relaxation of coronary arterial smooth muscle by glyceryl trinitrate, nitroprusside, nitrite and nitric oxide: effects of methylene blue and methemoglobin. J Pharmacol Exp Ther 1981;219:181-186.
29. Ignarro LJ, Lippton HL, Edwards JC, Baricos WFF, Hyman AL, et al. Mechanism of vascular smooth muscle by organic nitrates, nitrites, nitroprusside and nitric oxide: evidence for the involvement of S-nitrosothiols as active intermediates. J Pharmacol Exp Ther 1981;218: 739-749.
30. Ignarro LJ, Gruetter CA. Requirement of thiols for activation of coronary arterial guanylate cyclase by glyceryl trinitrate and sodium nitrite: possible involvement of S-nitrosothiols. Biochim Biophys Acta 1980;631:221-231.
31. Ignarro LJ, Kadowitz PJ. The pharmacological and physiological role of cyclic GMP in vascular smooth muscle relaxation. Annu Rev Pharmacol Toxicol 1985;25:171-191.
32. Williams PL, Warwick R, Dyson M, Bannister LH, eds. Gray's anatomy, 37th ed. New York: Churchill Livingston, 1989:685-690.
33. Moncada S, Gryglewski RJ, Bunting S, Vane JR. An enzyme isolated from arteries transforms prostaglandin endoperoxides to an unstable substance that inhibits platelet aggregation. Nature 1976;263:663-665.
34. Furchgott RF, Zawadzki JV. The obligatory role of endothelial cells in the relaxation of arterial smooth muscle by acetylcholine. Nature 1980;288:373-376.
35. Yanagisawa M, Kurihara H, Kimura S, Tomobe Y, Kobayashi M, Mitsui Y, et al. A novel potent vasoconstrictor peptide produced by vascular endothelial cells. Nature 1988;332:411-415.
36. Dinh-Xuan AT, Higenbottam TW. Non-prostanoid endothelium derived vasoactive factors. J Int Med Res 1989;17:305-315.
37. Griffith TM, Edward DH, Lewis MJ, Newby AC, Henderson AH. The nature of endothelium-derived vascular relaxant factor. Nature 1984;308:645-647.
38. Ignarro LJ, Buga GM, Wood KS, Byrns RE, Chaudhuri G. Endothelium-derived relaxing factor produced and released from artery and vein is nitric oxide. Proc Natl Acad Sci USA 1987;84:9265-9269.
39. Palmer RMJ, Ferrige AG, Moncada A. Nitric oxide release accounts for the biological activity of endothelium-derived relaxing factor. Nature 1987;327:524-526.
40. Cocks TM, Angus JA, Campbell JH, Campbell GR. Release and properties of endothelium-derived relaxing factor (EDRF) from endothelial cells in culture. J Cell Physiol 1985;123:310-320.
41. Furchgott RF. The role of the endothelium in the response of vascular smooth muscle to drugs. Annu Rev Pharmacol Toxicol 1984;24:175-197.
42. Furchgott RF. Studies on the relaxation of rabbit aorta by sodium nitrite: the basis for the proposal that the acid-activatable inhibitory factor from bovine retractor penis factor is inorganic nitric oxide and endothelium-derived relaxing factor is nitric oxide. In: Vanhoutle PM, ed. Vasodilatation, vascular smooth muscle, peptides, autonomic nerves and endothelium. New York: Raven Press, 1988:401-404.
43. Cremona G, Dinh-Xuan AT, Higenbottam TW. Review: endothelium-derived relaxing factor and the pulmonary circulation. Lung 1991;169:185-202.
44. Vane JR, Angaard EE, Botting RM. Regulatory functions of the vascular endothelium. N Engl J Med 1990; 323:27-36.
45. West JB. Respiratory physiology, 4th ed. Baltimore: Williams and Wilkins, 1990:43-44.
46. Harper RW. A guide to respiratory care: Phys. 1 Clin. Applic. Philadelphia: JB Lippincott Co, 1981.

47. West JB. Pulmonary pathophysiology, 4th ed. Baltimore: Williams & Wilkins, 1990:29.
48. Fishman AP. Hypoxia on the pulmonary circulation: how and where it acts. Circ Res 1976;38:221-231.
49. Philips JB III. Prostaglandin and related compounds in the perinatal pulmonary circulation. Pediatr Pharmacol 1983;4:129-142.
50. Edwards JC, Ignarro LJ, Hyman AL, Kadowitz PJ. Relaxation of intra-pulmonary artery and vein by nitrogen oxide-containing vasodilators and cyclic GMP. J Pharmacol Exp Ther 1984;228:33-42.
51. Holzmann S. Endothelium induced relaxation by acetylcholine associated with large rises in cyclic GMP in coronary arterial strips. J Cyclic Nucl Res 1982;8:409-419.
52. Melot C, Naeije R, Mols P, Vandenbossche JL, Denolin H. Effects of nifedipine on ventilation/perfusion matching in primary pulmonary hypertension. Chest 1983; 83:203-207.
53. Abu-Osba YK, Manasra K, Galal O, Rejjal A. Treatment of pulmonary hypertension of newborn (PPHN) with magnesium (abstract). Pediatr Res 1990;27(2): 294A.
54. Gottlieb SS, Wood LDH, Hansen DD, Long R. The effect of nitro-prusside on pulmonary edema, oxygen exchange, and blood flow in hydrochloric acid aspiration. Anesthesiology 1987;67:203-210.
55. Radermacher P, Huet Y, Pluskwa F, Herigault R, Mal H, Teisseire B, et al. Comparison of ketanserin and sodium nitroprusside in patients with severe ARDS. Anesthesiology 1988;68:152-157.
56. Radermacher P, Santak B, Becker H, Falke KJ. Prostaglandin E1 and nitroglycerin reduce pulmonary capillary pressure but worsen ventilation-perfusion distributions in patients with adult respiratory distress syndrome. Anesthesiology 1989;70:601-606.
57. Frostell C, Fratacci MD, Wain JC, Jones R, Zapol WM. Inhaled nitric oxide: a selective pulmonary vasodilator reversing hypoxic pulmonary vasoconstriction. Circulation 1991;83(6):2038-2047.
58. Davidson D, Eldemerdash A. Endothelium derived relaxing factor: presence in pulmonary and systemic arteries of the newborn guinea pig. Pediatr Res 1990;27 (2):128-132.
59. Abnan SH, Charfield BA, Hall SL, McMurtry IF. Role of endothelium-derived relaxing factor during transition of pulmonary circulation at birth. Am J Physiol 1990; 259:H1921-1927.
60. Fineman JR, Heymann MA, Soifer SJ. N omega-nitro-L-arginine attenuates endothelium-dependent pulmonary vasodilation in lambs. Am J Physiol 1991;260: H1299-1306.
61. Shaul PW, Farrar MA, Zellers TM. Oxygen modulates endothelium-derived relaxing factor production in fetal pulmonary arteries. Am J Physiol 1992;262:H355-364.
62. Tiktinsky MH, Cummings JJ, Morin FC III. Acetylcholine increased pulmonary blood flow in intact fetuses via endothelium-dependent vasodilation. Am J Physiol 1992;262:H406-410.
63. Ignarro LJ, Adams JB, Horwitz PM, Wood KS. Activation of soluble guanylate cyclase by NO-hemoproteins involves NO-heme exchange. Comparison of heme-containing and heme-deficient enzyme forms. J Biol Chem 1986;262(11):4997-5002.
64. Pollock JS, Forstermann U, Mitchell JA, et al. Purification and characterization of particulate endothelium-derived relaxing factor synthase from cultured and native bovine aortic endothelial cells. Proc Natl Acad Sci USA 1991;88:10480-10484.
65. Dinh-Xuan AT, Higenbottam J, Pepke-Zaba J, Butt AY, Cremona G. Evidence for dysfunction of the enzyme nitric oxide synthase in the mechanisms of impaired pulmonary endothelium-dependent relaxation in chronic hypoxic lung disease (abstract). Am Rev Respir Dis 1992;145(4):A207.
66. Moncada S, Palmer RMJ, Higgs EA. Biosynthesis of nitric oxide from L-arginine: a pathway for the regulation of cell function and communication. Biochem Pharmacol 1988;38:1709-1715.
67. Palmer RMJ, Ashton DS, Moncada S. Vascular endothelial cells synthesize nitric oxide from L-arginine. Nature 1988;333:664-666.
68. Eddahibi S, Carville C, Raffestin B, Adnot S. In vivo administration of L-arginine restores endothelium dependent relaxant activity in the pulmonary circulation of chronically hypoxic rats (abstract). Am Rev Respir Dis 1992;145(4):A208.
69. Schmidt HH, Nau H, Wittfoht W, Gerlach J, Prescher KE, Klein MM, et al. Arginine is a physiological precursor of endothelium-derived nitric oxide. Eur J Pharmacol 1988;54(2):213-216.
70. Griffith OW, Park KH, Levi R, Gross SS. The role of plasma arginine in nitric oxide synthesis: studies with arginase-treated rats. Second International Meeting on the Biology of Nitric Oxide. London, October, 1991.
71. Palmer RMJ, Moncada S. A novel citrulline-forming enzyme implicated in the formation of nitric oxide by vascular endothelial cells. Biochem Biophys Res Commun 1989;158:348-352.
72. Ignarro LJ, Gruetter CA, Hyman AL, Kadowitz PJ. Molecular mechanisms of vasodilation. In: Poste G, Crooke T, eds. Dopamine receptor agonist. New York: Plenum, 1983:259-288.
73. Lippton HL, Gruetter CA, Ignarro LJ, Meyer RL, Kadowitz PJ. Vasodilator actions of several N-nitroso compounds. Can J Physiol Pharmacol 1982;60:68-75.
74. Ignarro LJ, Burke TM, Wood KS, Wolin MS, Kadowitz PJ. Association between cyclic GMP accumulation and acetylcholine-elicited relaxation of bovine intrapulmonary artery. J Pharmacol Exp Ther 1983;228:682-690.
75. Rapoport RM, Murad F. Agonist-induced endothelium-dependent relaxation in rat thoracic aorta may be mediated through cGMP. Circ Res 1983;52:352-357.
76. Griffith TM, Edwards DH, Lewis MJ, Henderson AH. Evidence that cyclic guanosine monophosphate (cGMP)

mediates endothelium-dependent relaxation. Eur J Pharmacol 1985;112:195-202.
77. Macleod KM, Ng DDW, Harris KH, Diamond J. Evidence that cGMP is the mediator of endothelium-dependent inhibition of contractile responses of rat arteries to A-adrenoceptor stimulation. Mol Pharmacol 1987;32:59-64.
78. Feelisch M, Noack E. Nitric oxide (NO) formation from nitrovasodilators occurs independently of hemoglobin or non-heme iron. Eur J Pharmacol 1987;142(3):465-469.
79. Bigson QH, Roughton FJW. The kinetics of equilibria of the reactions of nitric oxide with sheep hemoglobin. J Physiol (Lond) 1957;136:507-526.
80. Oda H, Kusumoto S, Nakajimia T. Nitrosylhemoglobin formation in the blood of animals exposed to nitric oxide. Arch Environ Health 1975;30:453-465.
81. Chiodi H, Mohler JG. Effects of exposure of blood hemoglobin to nitric oxide. Environ Res 1985;37:355-363.
82. Ignarro LJ. Biological actions and properties of endothelium derived nitric oxide formed and released from artery and vein. Circ Res 1989;65:1-21.
83. Borland CD, Higenbottam TW. A simultaneous single breath measurement of pulmonary diffusing capacity with nitric oxide and carbon monoxide. Eur Respir J 1989;2(1):56-63.
84. Doyle MP, Hoekstra JW. Oxidation of nitrogen oxides by bound dioxygen in haemoproteins. J Inorg Biochem 1981;14:351-358.
85. Borland CDR, Harmes K, Cracknell N, Mack D, Higenbottam TW. Methaemoglobin levels in smokers and nonsmokers. Arch Environ Health 1985;40:330-333.
86. Norman V, Keith CH. Nitrogen oxides in tobacco smoke. Nature 1965;205:915-916.
87. Rossaint R, Falke KJ, Keitel M, Lopez F, Pison U, Slama K, et al. Inhaled nitric oxide in contrast to infused prostacyclin selectively reduces pulmonary hypertension and improves gas exchange in severe ARDS (abstract). Am Rev Respir Dis 1992;145(4):A185.
88. Falke KJ, Rossaint R, Keitel M, et al. Successful treatment of severe adult respiratory distress syndrome with nitric oxide: the first three patients. Second International Meeting on the Biology of Nitric Oxide, London, October, 1991.
89. Blomqvist H, Frostell C, Hedenstierna G, Zapol WM. Inhaled nitric oxide (NO): a selective pulmonary vasodilator reversing human hypoxic pulmonary vasoconstriction (HPV) (abstract). Circulation 1991;84: A361.
90. American Thoracic Society, American Lung Association. International Conference. Final Program, Miami Beach: ATS/ALA, 1992.
91. Pepke-Zaba J, Higenbottam TW, Dinh-Xuan AT, Stone D. Wallwork J. Inhaled nitric oxide as a cause of selective pulmonary vasodilatation in pulmonary hypertension. Lancet 1991;338:1173-1174.
92. Bigatello LM, Hurford WE, Kacmarek RM, Roberts JD Jr, Zapol WM. Inhaled nitric oxide is a selective pulmonary vasodilator in septic patients with severe ARDS (abstract). Am Rev Respir Dis 1992;145(4):A185.
93. Moinard J, Pillet O, Castaing Y, Guenard H. Inhaled nitric oxide: effects on pulmonary circulation and gas exchange in man (abstract). Am Rev Respir Dis 1992;145 (4):A207.
94. Devlin R, Horstman D, Becker S, Gerrity T, Madden M, Koren H. Inflammatory response in humans exposed to 2.0 ppm NO_2 (abstract). Am Rev Respir Dis 1990;145 (4):A456.
95. Gaston B, Drazen JM, Jansen A, Sugarbaker DA, Loscalzo J, Stamler JS. Relaxation of human airways invitro by S-nitrosothiols (abstract). Am Rev Respir Dis 1992;145(4):A383.
96. Clutton-Brock J. Two cases of poisoning by contamination of nitrous oxide with higher oxides of nitrogen during anaesthesia. Br J Anaesth 1967;39:388-392.
97. Austin AT. The chemistry of the higher oxides of nitrogen as related to the manufacture, storage and administration of nitrous oxide. Br J Anaesth 1967;39: 345-350.
98. Greenbaum R, Bay J, Hargreaves MD, Kain ML, Kelman GR, Nunn JF, et al. Effects of higher oxides of nitrogen on the anesthetized dog. Br J Anaesth 1967;39: 393-404.
99. Sackheim GI, Lehman DD, eds. Chemistry for the health sciences. New York: MacMillan, 1990:130-133.
100. Brown RF, Clifford WE, Marrs TC, Cox RA. The histopathology of rat lung following short term exposures to mixed oxides of nitrogen (NOx). Br J Exp Pathol 1983;64(6):579-593.
101. Samet JM, Utell MJ. The risk of nitrogen dioxide: what have we learned from epidemiological and clinical studies? Toxicol Indus Health 1990;6(20):247-262.
102. Stavert DM, Lehnert BE. Nitrogen oxide and nitrogen dioxide as inducers of acute pulmonary injury when inhaled at relatively high concentrations for brief periods. Inhal Toxicol 1990;2:53-57.
103. MMWR August 26, 1988, Vol 37, NO S-7(21).
104. Clyne MAA, Thrush BA, Wayne RP. Trans Faraday Soc 1964;60:359.
105. Seitz WR, Neary MP. Chemiluminescence and bioluminescence. Contemp Topics Anal Clin Chem 1977; 1:49-125.
106. Kricka LJ, Carter TJN, eds. Clinical and biochemical luminescence. London: Marcel-Dekker, 1982:135-136.
107. Fontijn A, Sabadell AJ, Ronco RJ. Homogeneous chemiluminescent measurement of nitric oxide with ozone. Anal Chem 1970;42(6):574-579.
108. Pai GT, Payne WJ, LeGall J. Use of a chemiluminescence detector for quantitation of nitric oxide produced in assays of denitifying enzymes. Anal Biochem 1987;166:150-157.
109. Kacmarek RM. Respiratory care practitioner: carpe diem! Respir Care 1992;37(3):264-269.

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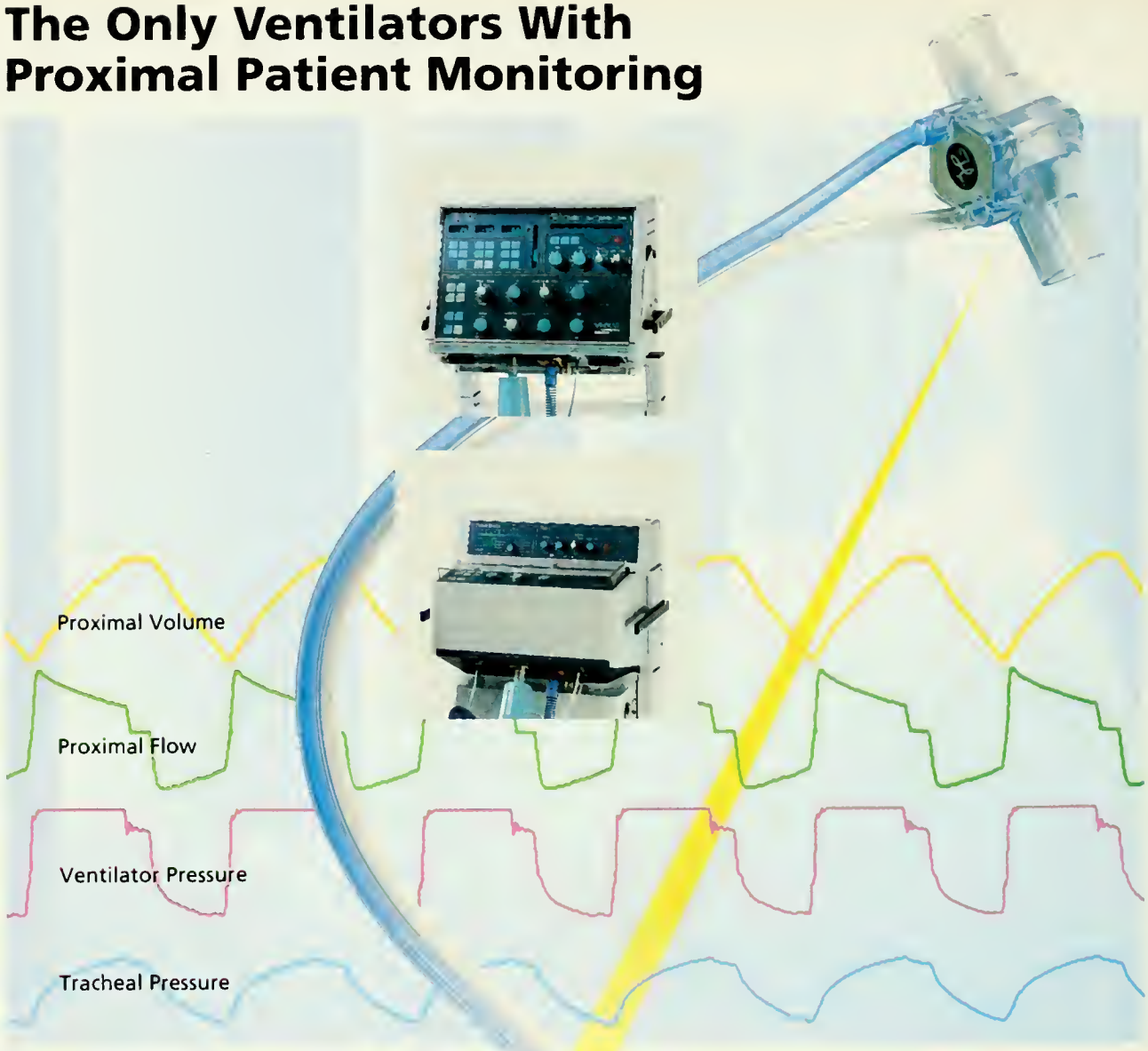

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PFT Corner #45— Asthma and the Vocal Cord Dysfunction Syndrome: When Wheezing Is Hysterical

Jack Wanger RPFT RRT and William R Beam MD

A 36-year-old woman with a complex medical and personal history was admitted to our facility for evaluation. She had been a licensed practical nurse (LPN) for 12 years and reported that she had come from a dysfunctional family. About two years prior to admission, she had experienced an apparent episode of anaphylaxis after ingesting a nonsteroidal anti-inflammatory medication and had required intubation and mechanical ventilation. Subsequently, she was hospitalized three times within the next 2 months for bronchospasm. Over the next 2 months she had been tapered off of all asthma medication except a beta agonist via metered dose inhaler to be used as needed. She had done well for several months until she suffered another apparent respiratory arrest requiring intubation. This episode was preceded by the ingestion of approximately 6 oz of red wine and, consequently, was thought to represent an adverse reaction to sulfites in the wine. During the next 8 months, she required eight hospitalizations and numerous emergency room visits for refractory asthma and episodes of bronchospasm. Due to the refractory nature of her symptoms, she was said to have steroid-resistant asthma and was treated with a protocol combining troleandomycin (TAO) and methylprednisolone and later with methotrexate, in an attempt to reduce the corticosteroids being used to treat her symptoms. During attempts to reduce her corticosteroid dosage, she required two hospitalizations for increasing bronchospasm and suffered another respiratory arrest after ingesting chocolate, again requiring intubation and mechanical ventilation. However, a fiberoptic evaluation of her airways following extubation revealed vocal-cord mucus stranding and laryngeal dyskinesia suggestive of the vocal cord dysfunction syndrome. The patient rejected this as a

Table 1. Results of Pulmonary Function Tests Performed on Admission before and after Albuterol by a 36-Year-Old Woman

Test	Before Bronchodilator	After Bronchodilator
TLC (L)	5.24 (94)*	5.46
TGV (L)	2.68 (87)	2.17
RV (L)	1.54 (113)	1.27
FVC (L)	4.19 (100)	4.59
FEV_1 (L)	3.00 (85)	3.36
FEV_1/FVC (%)	75	73
FEF_{50}/FIF_{50}†	1.80	1.75
D_{LCO} (mL · torr · min^{-1})	35.30 (114)	

* Values in parentheses are percent predicted.

† FEF_{50} = forced expiratory flow at 50% of vital capacity; FIF_{50} = forced inspiratory flow at 50% of vital capacity.

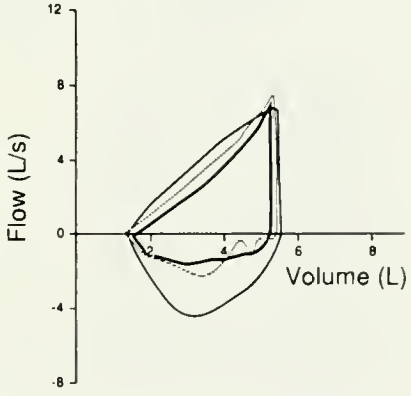

Fig. 1. The flow-volume relationships for a 36-year-old woman with asthmatic symptoms. The light solid line is the reference (predicted) relationship; the dark solid line is the flow-volume loop prior to inhaled albuterol; and the dotted line is the flow-volume loop after albuterol.

Mr Wanger is Manager and Technical Director, Pulmonary Physiology Unit; Dr Beam is Staff Physician and Instructor of Medicine—National Jewish Center for Immunology and Respiratory Medicine, Denver, Colorado.

diagnosis and sought another medical opinion from our institution.

On admission to our hospital, she was taking TAO/methylprednisolone, methotrexate, and theophylline orally, and atropine, albuterol, cromolyn sodium, and triamcinalone by inhalation. Her chest radiograph revealed normal lung volumes without cardiovascular abnormality, no evidence of adenopathy, and mildly prominent bronchovascular markings. These findings were consistent with the diagnosis of asthma. Her admission pulmonary function test results are shown in Table 1 and Figure 1.

Questions

1. What is your interpretation of the results of admission testing?
2. What additional tests do you recommend?

Answers and Discussion

1. Admission PFTs: The admission PFTs show normal lung volumes and mild airflow limitation, which improved after inhaled albuterol. The inspiratory portions of the flow-volume loops are flattened. The D_{LCO} is within normal limits.

2. Additional Tests: The diagnosis of vocal cord dysfunction had been previously postulated. However, to document this phenomenon a laryngoscopy during symptoms or following an abnormal flow-volume loop was indicated. Whether our patient also has asthma remained unclear. Therefore, testing for hyperreactive airways was indicated (eg, histamine or methacholine provocation challenge). If our patient does have hyperresponsive airways, it may also be useful to determine if sulfites are a trigger.

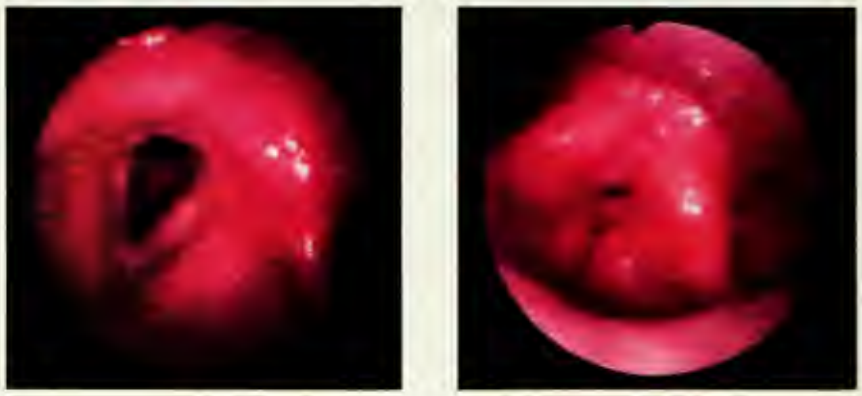

Fig. 2. Two views of the glottis during fiberoptic laryngoscopy in a 36-year-old woman with vocal cord dysfunction. On the left, the vocal cords are seen during quiet tidal breathing. On the right, an almost complete anterior closure of the airway is seen during forced respiration with the pathognomonic posterior chink clearly evident.

Results of Additional Tests

The results of the direct fiberoptic laryngoscopy revealed both inspiratory and expiratory vocal cord adduction sporadically during quiet breathing. The phenomenon worsened with forced inspirations (Fig. 2). Additionally, a posterior chink was evident with forced maneuvers. No nasal polyps or laryngeal masses were seen.

The results of the histamine provocation test are shown in Table 2 and Figure 3. The technician noted that the patient had increasing difficulty during inspiration with the higher doses of histamine. The interpretation of this test was mild airway hyperresponsiveness to histamine with a PC_{20} FEV_1 of 5.0 mg/mL.

A metabisulfite provocation test was subsequently performed using 5, 25, 50, and 100 mg/mL doses of metabisulfite administered orally. Pulmonary function tests were performed at 5 and 30 minutes after each dose. The results showed a negative response.

Discussion

The case described in this Corner is typical of a group of patients with the diagnosis of vocal cord dysfunction (VCD) syndrome in that the symptoms of bronchospasm seem out of proportion to the severity of asthma quantitated by objective testing. These patients are difficult to treat and frequently have side effects from their treatment (eg, Cushing's syndrome).

Historically, patients with VCD syndrome have been described as having Munchausen's stridor,[1] factitious asthma,[2] emotional wheezing,[3] and psychogenic pharyngeal constriction.[4] The clinical features described

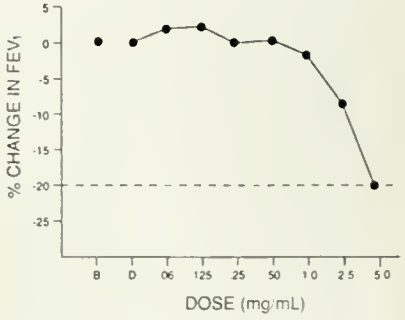

Fig. 3. The graphic representation of the histamine provocation test (challenge). The % change in FEV_1 is calculated from the FEV_1 measured after inhalation of the diluent (D). The dosing begins at 0.06 mg/mL and increases in doubling increments up to 5.0 mg/mL. A 20% fall in FEV_1 was observed after inhalation of the 5.0 mg/mL dose. The PC_{20} FEV_1 is 5.00 mg/mL.

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Table 2. Results of Histamine Bronchial Provocation Test in a 36-Year-Old Woman with a Complex History

Dose (mg/mL)	FEV_1 (L)	% Change*
Baseline	3.36	—
Diluent	3.32	—
0.06	3.37	+2
0.125	3.40	+2
0.25	3.34	0
0.50	3.32	0
1.00	3.27	−2
2.50	3.03	−9
5.00†	2.63	−20

* Percent change calculated from FEV_1 measured after administration of diluent.
† PC_{20} FEV_1 = 5.0 mg/mL.

differ somewhat but can be termed inspiratory VCD, expiratory VCD, or inspiratory and expiratory VCD.[5]

The diagnostic evaluation of patients with presumed asthma and VCD requires care. The history may give valuable clues, directing the physician to vocal cord dysfunction.[6] Many so-called asthmatics will point to the area of the vocal cords when asked where they believe the airflow limitation is occurring.

The patient population most commonly diagnosed with VCD syndrome is female between the ages of 20 and 40 years. Also, previous medical or paramedical work experience is commonly reported.[6] Auscultation of the laryngeal area is not always a reliable test. Stridor can occur with normal vocal cord function, and conversely there can be VCD and no stridor.

The pulmonary function laboratory data are usually (and remarkably) variable. When VCD patients are asymptomatic, lung volumes, spirometry, and arterial blood gases are normal.

When symptomatic, the VCD patient usually demonstrates an abnormal flow-volume loop. The inspiratory portion or both inspiratory and expiratory portions may be abnormal. Coughing or panting will sometimes relieve the abnormal adduction of the cords, and flow will be increased in the expiratory portion. Thus, the flow-volume loop may be helpful in suggesting the VCD syndrome but is not diagnostic.[6]

Arterial blood gas values usually remain normal even during episodes of wheezing and dyspnea.[7] However, hypoxemia and hypercapnia[5,6] can occur and make the clinical decision to intubate a patient a difficult one if the diagnosis of VCD has not yet been firmly established. A clue to the diagnosis of VCD following intubation may be the lack of elevated peak airway pressures during mechanical ventilation.

The definitive diagnostic test is direct fiberoptic laryngoscopy.[6] This procedure should be performed during an acute exacerbation. Panting, fast inspiration, and phonation should be included in the maneuver. Again, coughing and panting may reverse the abnormal vocal cord movement. Occasionally direct stimulation of the vocal cords with the laryngoscope uncovers the dysfunction, but this is not routinely recommended because it may result in reflex laryngospasm and produce a false-positive evaluation.

The most common dysfunctional pattern is adduction of the anterior two thirds of the vocal cords with a posterior diamond-shaped chink. While this pattern usually occurs during inspiration, it can also be present during the entire respiratory cycle.

The VCD syndrome may be classified as a variation of a hysterical conversion disorder. The range of psychopathology associated with VCD includes depression, obsessive-compulsive personality, passive-dependent personality, and adjustment reaction disorders. A majority of VCD patients are single and often have had difficulty emancipating themselves from childlike relationships with their families. However, VCD patients do not consciously control the process, and therefore VCD is not a malingering disorder.[6] The treatment of VCD is best approached with a multidisciplinary team consisting of the physician, psychiatrist, and speech therapist. The approach used when informing patients they have VCD may affect their response and, thus, it should be done with care.[6]

The speech therapy techniques used to treat patients with VCD syndrome are adapted from those techniques used for patients with other functional voice disorders (eg, hoarseness, laryngitis). Patients are taught to focus attention away from the larynx and the inspiratory phase of breathing, to relax the oropharyngeal muscle groups, and to concentrate on active expiration using the abdominal muscles.

In our patient, VCD syndrome was the primary cause of her pulmonary disability. However, it also appears that she has mild airway hyperreactivity. She has improved significantly after numerous speech therapy sessions, and is again maintained on a beta-agonist MDI only as needed.

In conclusion, VCD syndrome is an uncommon cause of wheezing. It should be suspected in patients with

ecurrent acute episodes of wheezing iat do not respond to usual asthma eatment except intubation. How-ver, as with our case, VCD can oc-ur in conjunction with bronchial hy-erreactivity, and a systematic diag-ostic approach is needed to es-ıblish the most likely source of the atient's symptoms.

REFERENCES

1. Patterson R, Schatz M, Horton M. Munchausen's stridor: non-organic laryngeal obstruction. Clin Allergy 1974;4:307-310.
2. Downing ET, Braman SS, Fox MJ, Corrao WM. Factitious asthma: physiological approach to diagnosis. JAMA 1982;248:2878-2881.
3. Rodenstein DO, Francis C, Stanescu DC. Emotional laryngeal wheezing: a new syndrome. Am Rev Respir Dis 1983;127:354-356.
4. Nagai A, Yamaguchi E, Sakamoto K, Takahashi E. Functional upper airway obstruction: psychogenic pharyngeal constriction. Chest 1992; 101:1460-1461.
5. Goldman J, Muers M. Vocal cord dysfunction and wheezing (editorial). Thorax 1991;46:401-404.
6. Martin RJ, Blager FB, Gay ML, Wood RP. Paradoxic vocal cord motion in presumed asthmatics. Semin Respir Med 1987;8:332-337.
7. Christopher KL, Wood RP II, Eckert RC, Blager FB, Raney RA, Souhrada JF. Vocal-cord dysfunction presenting as asthma. N Engl J Med 1983;308:1566-1570.

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Is This a Simple Case of Respiratory Distress Syndrome?

Katherine A Douglas RRT and Kathleen S Carlson MD

A 25-year-old woman who was in her sixth pregnancy (gravida VI) had given birth to 4 children (para IV), and had lost 1 nonviable fetus (abortus I), was admitted to the hospital for suspected labor at 32-weeks gestation. A questionable fetal abnormality suggesting dwarfism had been documented by ultrasound earlier in the pregnancy; however, there was no family history of birth defects or dwarfism. The mother used alcohol infrequently and was a one-half pack per day smoker. The only medications she had used during the pregnancy had been stool softeners, terbutaline, and vitamins.

After a spontaneous vaginal, vertex delivery, the 1820-g boy cried spontaneously but quickly developed increasing respiratory distress and bradycardia unresponsive to bag and mask ventilation with 100% oxygen. The infant was intubated and ventilated, and pharmacologic support via arterial and venous catheters was initiated. Apgar scores were 4 and 7 at 1 and 5 minutes, respectively.

On physical exam, the infant's small chest diameter and very short arms were noted. Breath sounds were coarse; no heart murmur was heard on auscultation. Bowel sounds were present. The testes were not descended. The skull was very soft; the anterior fontanel could not be appreciated. Radiographs were obtained; the chest film appears in Figure 1.

Ms Douglas is the NICU orientor with the Department of Respiratory Care and Dr Carlson is associated with the Division of Neonatology of The Toledo Hospital's Reuben Center for Women and Children. Dr Carlson is also clinical assistant professor in the Department of Pediatrics, Medical College of Ohio at Toledo, Toledo, Ohio.

Fig. 1. Chest radiograph of a newborn infant with respiratory distress and bradycardia unresponsive to bag-mask ventilation with 100% oxygen.

Questions

Radiographic Findings: Aside from the pulmonary parenchyma, what are the most remarkable features of the chest radiograph?

Tube Position: Is the endotracheal tube in proper position?

Answers and Discussion on Next Page

Answers

Radiographic Findings: The chest film is particularly notable for the absence of tracheal rings, the absence of ossification of the cervical and thoracic vertebrae, and the presence of short, irregular, and poorly developed ribs.

Tube Position: The endotracheal tube is high at the clavicular level and should be advanced one centimeter.

Discussion

The poor ossification shown in the chest film and in the initial skull film shown here in Figure 2 is indicative of one of the following pathologies: Achondrogenesis Type I, Achondrogenesis Type II, or a severe form of hypophosphatasia. In this infant, the diagnosis of infantile hypophosphatasia was made from the presence of serum alkaline phosphatase levels of 4-11 IU/L (normal is 50-273 IU/L) and urinary excretion of phosphoethanolamine of 10,290 μmol/g creatinine (normal 83-200 μmol/g). Blood for chromosome studies was drawn from the patient, and results were normal.

Fig. 2. Skull film of the newborn infant whose chest film is shown in Figure 1.

Hypophosphatasia, a rare metabolic disorder estimated to occur once in 100,000 births,[1] is most commonly due to the presence of an autosomal recessive trait, although an autosomal dominant form has been described.[2]

In congenital hypophosphatasia, the affected infant presents at birth with skeletal deformities and respiratory distress.[3] The condition is usually lethal during early infancy, with respiratory insufficiency the usual cause of death.[4] (A less severe form of hypophosphatasia can manifest as an osteomalacia in adults). Diagnosis is made by confirming very low levels of serum alkaline phosphatase and excessive urinary secretion of phosphoethanolamine.

Radiographs of affected infants are notable for poor ossification of the cranium, wide sutures, poorly ossified long bones with irregular lack of metaphyseal mineralization, bowed lower extremities, and short ribs with rachitic changes.[4-6] All these findings were noted in our patient.

In these infants, growth cartilage is of abnormal structure with long, irregular columns of hypertrophic cells, only a few visible areas of calcification, and excessive proliferation of osteoid tissue.[3]

Prenatal diagnosis of hypophosphatasia is possible using an alkaline phosphatase assay following chorionic villus sampling in the first trimester.[7] Mid-trimester prenatal diagnosis using ultrasound has also been reported. No effective therapy is currently available for hypophosphatasia, with attempts at treatment with a low calcium diet, steroids, and vitamin K proving ineffective.[4]

In spite of increasing ventilatory support, our patient developed progressive respiratory failure. The parents elected to remove the baby from the ventilator on the 22nd day of life; he succumbed within the hour. An autopsy was refused.

REFERENCES

1. Whyte MP. Hypophosphatasia. In: Scriver CR, Beaudet AL, Sly WS, Valle D, eds. The metabolic basis of inherited disease. New York: McGraw-Hill, 1989:2843-2856.
2. Smith DW. Hypophosphatasia syndrome. In: James KL, ed. Smith's recognizable patterns of human malformation. Philadelphia: WB Saunders, 1988:346-347.
3. Borochowitz Z, Rimoin L. Skeletal pathology: the congenital chondrodysplasias. In: Reed GB, Claireaux AE,

Bain AD, eds. Diseases of the fetus and newborn: pathology, radiology and genetics. St Louis: CV Mosby Co, 1989:497-514.
4. Wolfish NM, Heick H. Hyperparathyroidism and infantile hypophosphatasia: effect of prednisone and vitamin K therapy. J Pediatr 1979;95:1079-1081.
5. Rathbun JC. Hypophosphatasia: a new developmental anomaly. Am J Dis Child 1948;75:822-823.
6. MacPherson RI, Kroeker M, Houston CS. Hypophosphatasia. J Can Assoc Radiol 1972;23:16-26.
7. Warren RC, McKenzie CF, Rodeck CH, Moscoso G, Brock DJ, Barron L. First trimester diagnosis of hypophosphatasia with a monoclonal antibody to the liver/bone/kidney isoenzyme of alkaline phosphatase. Lancet 1985;2:856-858.

CORRECTIONS

The following are corrections to two Clinical Practice Guidelines published in the August issue (pages 918 and 908, respectively). They have been prepared so that you can (*and we encourage you to*) cut them out and paste or tape them over the incorrect copy. We regret the errors.

OT-CC 4.0 INDICATIONS:

Documented hypoxemia: In adults, children and infants older than 28 days: (1) $P_{aO_2} \leq 55$ torr or $S_{aO_2} \leq 88\%$ in subjects breathing room air,[1,2] or (2) P_{aO_2} of 56-59 torr or S_{aO_2} or $S_{pO_2} \leq 89\%$ in association with specific clinical conditions (eg, cor pulmonale, congestive heart failure, or erythrocythemia with hematocrit > 56).[3,4]

7.1.1.1.5 Reduced ear perfusion associated with heavy exercise has been shown to affect S_{pO_2}.[10,35-37]

The following is from Hess and Eitel, Monitoring during Resuscitation, Respir Care 1992;37(7):739-765. They have been prepared so that you can (*and we encourage you to*) cut them out and paste or tape them over the incorrect copy. (The first paragraph is found on Page 751.) We regret the errors.

The effect of hemorrhage on P_{cjO_2} has also been reported.[140-144] In a study of 16 normotensive emergency department patients with histories consistent with important blood loss, it was found that a conjunctival index ≤ 0.50 was always associated with a blood volume reduced by ≥ 15% below predicted values (Fig. 23).[140] In dogs, it has been shown that

Fig. 33. Survival from in-hospital resuscitation from 1961 to 1991. Dark boxes = survived arrest; hatched boxes = survived to hospital discharge.

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"Murder Mystery" for Student Practice of Pulmonary Physiology Calculations

Michael B Maron PhD and Frank J Bosso PhD

In the study of pulmonary physiology, there are a number of important calculations that can be made that can give insight into how well the lungs are functioning. These include calculations of alveolar partial pressure of O_2 (P_{O_2}), arterial O_2 content, physiological dead space, and alveolar ventilation. In the relatively short time available for the discussion of each organ system in medical physiology courses, students are often overwhelmed by the number of equations in the pulmonary unit. Although students are given the opportunity to practice doing these calculations in the form of problem sets, there is often a lack of interest in spending the time required to make these a useful exercise. In an attempt to make these problems more interesting, we have developed a "murder mystery" in which students are required to apply basic principles learned in the pulmonary unit to determine the identity of the murderer. To solve the case, students must calculate arterial O_2 content, O_2 delivery, physiological dead space, dead space ventilation, alveolar ventilation, and alveolar P_{O_2} and partial pressure of CO_2 (P_{CO_2}). The title of the exercise is "The Case of the Murdered Rapper: A Whodunit Where Basic Principles of Respiratory Physiology Are Brought To Bear Upon the Matter of the Identity of the Murderer." Although the scenario is admittedly farfetched, we have observed that its improbability is the critical ingredient that makes the exercise appealing.

Dr Maron and Dr Bosso teach in the Department of Physiology, Northeastern Ohio Universities College of Medicine, Rootstown, Ohio.

At the suggestion of Neil MacIntyre MD, Duke University Medical Center, Durham, North Carolina, this paper is reprinted, with slight modifications and with permission, from the American Journal of Physiology, 261 (Adv Physiol Educ 6):S3-S6, 1991. Educators wishing to use this paper for their students may photocopy it.

CAST OF CHARACTERS

Davenport Deadspace. The powerful chairman of a large international conglomerate (Environrape Inc) that has made billions but whose products have contributed to the defoliation of the Amazon rain forests.

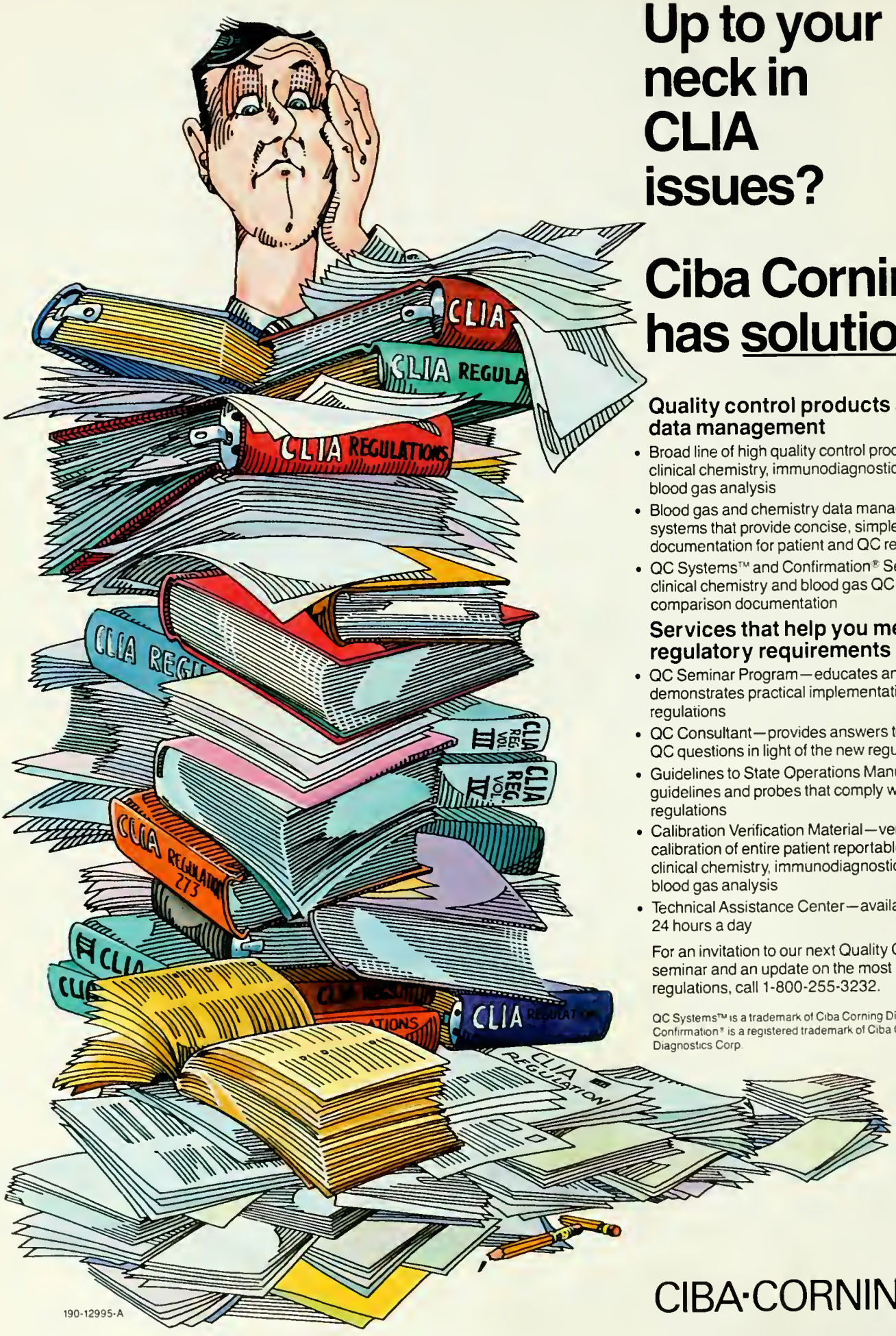
Daphne Deadspace. The lovely and beguiling daughter of Davenport Deadspace. A debutante of the first rank and heiress who is betrothed to Bobby Bronchus, a heavy metal rapper. She sings weekends with the Metropolitan Opera.

Bobby Bronchus. A heavy metal rapper, whose band (Bobby Bronchus and the Bronchial Tubes) is the rage of two continents and discrete sections of a third. Bobby channels much of his rapper earnings into causes advocating the preservation of the environment and is a member of 37 different environmental organizations. He is torn between his love for Daphne and that for the environment.

Victor Ventilation. The Deadspaces' loyal butler, who has worked for the family for the last 30 years. Generations of Ventilations have worked for generations of Deadspaces.

Aldo Alveolus PhD. A respiratory physiologist, who early in his career was an advocate of the O_2 secretion hypothesis of O_2 transfer from air to blood. His research is funded by grants from Enrivonrape Inc.

Madeline Medstudent. A medical student, who is working her way through medical school as a part-time employee of the Acme Detective Service. She has been assigned to solve the murder.



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THE MURDER

Bobby Bronchus has volunteered to be a subject in one of Dr Alveolus's lung function studies and will have his CO diffusion capacity measured. The amount of CO used in this test is normally very small and poses no threat to life. Someone, however, has switched the gas cylinders, and the new tank now contains a lethal concentration of CO. Dr Alveolus's technician administers the test to Bobby, who quickly collapses and dies.

THE INVESTIGATION

Madeline arrives to investigate the death and immediately suspects foul play when she learns that CO has been used. The immediate question is, however, Was the CO concentration high enough to kill Bobby or did he die of natural causes? Madeline draws a sample of Bobby's arterial blood and finds that the hemoglobin concentration is 14.0 g/dL, and the carboxyhemoglobin level is 80%. Madeline then assumes that, at rest, Bobby would have had a cardiac output of ~ 5 L blood/min, an O_2 consumption of ~ 250 mL O_2/min, and an arterial P_{O_2} of ~ 100 torr. She quickly uses the Fick principle (see Appendix for formulas) to calculate Bobby's normal arteriovenous O_2 content difference at rest and then compares this value with his calculated arterial O_2 content to determine if the amount of CO found in Bobby's blood was enough to prevent normal O_2 delivery. Madeline reasons that the arteriovenous O_2 content difference at resting cardiac output and O_2 consumption reflects the amount of O_2 that must leave the blood to maintain a normal resting metabolism. A finding that Bobby's blood was incapable of carrying even this amount would suggest that Bobby died a hypoxic death. *Perform these calculations and fill in the blanks in Table 1 to determine if Bobby died of CO poisoning.*

Table 1. Calculations To Determine CO Poisoning

Arteriovenous oxygen content difference	?	mL O_2/dL
Arterial oxygen content in presence of 80% carboxyhemoglobin	?	mL O_2/dL

Calculate the missing values. Did Bobby die of CO poisoning?

The question now becomes, "Whodunit?" Madeline quickly learns that all of the characters have motives to commit the murder. Davenport has learned that Bobby has found out about the rape of the Amazon rain forests and will soon be going public with the information. Davenport thus sees a long prison sentence ahead and the evaporation of the pretentious life style that he has come to love. Daphne has several motives. First, she loves her father and cannot bear to see him suffer. Second, she too has come to enjoy the lifestyle made possible by her father's ill-gotten riches and would not be able to continue her voice lessons without this support. Finally, she has recently learned that Bobby has been having an affair with the Bronchial Tubes' female drummer, Helen Hemoglobin. Victor, the butler, has a motive in that his loyalty to the Deadspace family could drive him to commit any manner of heinous wrongdoing to protect the Deadspace family. Finally, Dr Alveolus stands to lose his research support if Environape Inc experiences financial difficulties.

Madeline further learns that the CO cylinder could have been switched only between 2:00 and 2:45 PM on the preceding Saturday. All of the suspects were attending a lawn party at the Deadspace mansion at that time and all have alibis except for this 45-min period. None of the suspects can establish exactly where they were during this time. Madeline realizes that the murderer would have had to run from the mansion to the lab (a distance of ~ 1 mile), exchange the cylinder, and then run back to the mansion during this period. Madeline figures that the murderer would have to complete each leg of the run in ~ 10 min. She does a quick mental calculation and concludes that the murderer would be required to deliver ~ 2,300 mL O_2/min to the working muscles to accomplish this feat in the allotted time. As a first step, she has arterial blood samples drawn from each of the suspects and also has their maximal cardiac outputs determined on a treadmill. She uses these data to calculate each individual's O_2 content and maximal O_2 delivery rate and is quickly able to eliminate one of the suspects. *Perform these calculations and tabulate them in Table 2 to determine which suspect Madeline is able to eliminate.*

Back at school, Madeline attends an exercise physiology lecture and learns that few individuals

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Table 2. Arterial O_2 Content and Maximal O_2 Delivery Rate Values

Suspects	[Hb]* (g/dL)	P_{aO_2} (torr)	Sat (%)	$\dot{Q}$ (L/min)	C_{aO_2} (mL/dL)	Max Del (mL/min)
Davenport	15	97	98	20	?	?
Daphne	12	95	97	16	?	?
Victor	8	98	98	30	?	?
Aldo	8	70	90	12	?	?

*[Hb] = arterial hemoglobin concentration; P_{aO_2} = arterial partial pressure of O_2; Sat = arterial HbO_2 saturation; $\dot{Q}$ = maximal cardiac output; C_{aO_2} = arterial O_2 content; Max Del = maximal rate of O_2 delivery. *Calculate the values for C_{aO_2} and maximal delivery.*

These data eliminate _____?_____ as the suspect.

are able to maintain their maximal rate of O_2 delivery for very long. Thinking about this, she reasons that none of the suspects would be able to maintain an O_2 delivery rate > 75% of maximum. A lightning-quick series of calculations immediately eliminates another suspect. *Perform these calculations and enter the results in Table 3 to find out who is eliminated.*

Table 3. Maximal and 75% Maximal O_2 Delivery Rate Values

Suspects	Maximal Delivery (mL O_2/min)	75% Maximal (mL O_2/min)
Davenport	?	?
Daphne	?	?
Victor	?	?
Aldo	?	?

Note: Remember Madeline has calculated that an O_2 delivery of 2,300 mL/min is required to make the run in time. *Calculate the values for maximal delivery and 75% of maximal.*

These data eliminate _____?_____ as a suspect.

A few days later, Madeline returns to the scene of the crime to search for further clues. She interviews the cleaning crew and learns that they saw no one in the lab when they cleaned between 2:10 and 2:35 PM. Obviously, the murderer had hidden in the lab during this interval. All of the obvious places to hide, however, are filled with Dr Alveolus's experimental records dating back to the 1930s. On further inspection of the laboratory, Madeline finds Dr Alveolus's underwater weighing pool, a large tank built into the ground that is used to weigh subjects underwater for the determination of body fat content. By the pool, she finds a large piece of plastic tubing of the type used for plumbing. It is clear that the murderer had used the tubing as a snorkel while hiding from the cleaning crew in the underwater weighing pool.

Madeline determines the volume of the tubing by water displacement to be 135 mL. "This means that the murderer's dead-space ventilation increased while hiding in the tank," Madeline concludes. "This will have important implications for O_2 and CO_2 homeostasis. Perhaps the murder can be solved on this basis."

Madeline observes that the tubing is equipped with a pneumotach (a device that measures airflow) and has cable connections to an analog-to-digital converter attached to Dr Alveolus's computer. The tubing is obviously a homemade device for studies of dead space. Madeline turns on the computer and learns that the system was on during the time of the murder and that there are actual recordings of ventilation during the time the murderer was using the tubing as a snorkel. The records indicate that the total minute ventilation of the murderer while breathing through the tubing was 6,450 mL/min. Madeline realizes that all she has to do is find which suspect would have a total ventilation of 6,450 mL/min while breathing through the tubing at the bottom of the pool. This individual would have to be the murderer.

The most direct approach to solving this problem would be to measure the total ventilation of each suspect breathing through the tubing. Madeline is concerned, however, that the murderer would quickly understand what she is up to and would hyperventilate or hypoventilate during the test to change ventilation. Dejected, she continues to search through the computer records and finds

Table 4. Ventilatory Calculations

	Davenport	Daphne	Victor	Aldo
Control conditions				
Mixed expired P_{CO_2} (torr)	27	29	23.6	22
Arterial (= alveolar) P_{CO_2} (torr)	40	39	40	38
Tidal volume (mL)	510	450	487	400
Physiologic dead space (mL)	?	?	?	?
Total ventilation (mL/min)	5,100	5,400	5,844	6,000
Respiratory rate (breaths/min)	10	12	12	15
Dead-space ventilation (mL/min)	?	?	?	?
Alveolar ventilation (mL/min)	?	?	?	?
Added dead space (note that respiratory rate remains constant)				
Snorkel + physiologic dead space (mL)	?	?	?	?
Dead-space ventilation (mL/min)	?	?	?	?
Total ventilation (mL/min)	?	?	?	?

Calculate the missing values. Who is the murderer?

experimental data for all of the subjects from a study on dead space conducted by Dr Alveolus. "Eureka!" she shouts. Madeline has found mixed expiratory P_{CO_2}, arterial P_{CO_2}, tidal volume, total ventilation, and respiratory rate data under baseline conditions for each subject. Her strategy is now immediately clear. She reasons that she can use this information to calculate each suspect's normal alveolar ventilation and that this value should be similar (if the individual is to maintain CO_2 and O_2 homeostasis) under conditions of snorkel breathing. All she has to do is add the total dead-space ventilation (physiological + snorkel dead space) to this value to obtain the total ventilation. *Perform the calculations indicated in Table 4 to determine the identity of the murderer.*

It is now clear that _____?_____ is the murderer. Impressed and frustrated by Madeline's relentless logic, he cries, "Curses, done in by a medical student with a sound knowledge of respiratory physiology!" Before leaving the crime scene and turning her attention to biochemistry, Madeline decides to perform another series of calculations for relaxation. She knows from her pulmonary physiology lectures that to maintain CO_2 and O_2 homeostasis during snorkel breathing, one must maintain alveolar ventilation by increasing total ventilation. Madeline wonders what each of the suspect's alveolar P_{O_2} and P_{CO_2} would be if ventilation were not increased. *Help Madeline unwind by performing the calculations indicated in Table 5.*

Table 5. Determinations of Alveolar P_{O_2} and P_{CO_2} Values

	Davenport	Daphne	Victor	Aldo
Total ventilation under control conditions (mL/min)	5,100	5,400	5,844	6,000
Dead-space ventilation with added dead space (mL/min)	?	?	?	?
Alveolar ventilation (mL/min)	?	?	?	?
Alveolar P_{CO_2} (torr)	?	?	?	?
Alveolar P_{O_2} (torr)	?	?	?	?

Calculate the missing values.

APPENDIX

Symbols, constants, and equations are from West.[1]

Environmental Conditions and Constants

Barometric pressure (P_B) = 740 torr.
Water vapor pressure (P_{H_2O}) = 47 torr at 37°C.
Hemoglobin (Hb)-O_2 combining capacity = 1.39 mL O_2/g Hb.
O_2 solubility coefficient = 0.003 mL O_2/dL/torr.
Inspired O_2 fraction (F_{IO_2}) = 0.2093.
Respiratory exchange ratio (R) = 0.8.

Equations (in order of use)

Arteriovenous O_2 content difference (A-V_{O_2}) (Fick equation)

$$A\text{–}VO_2 = \dot{V}_{O_2}/\dot{Q} \qquad [A1]$$

where $\dot{Q}$ is cardiac output and $\dot{V}_{O_2}$ is the rate of O_2 consumption.

$$HbO_2 = (Hb)(HbO_2 \text{ combining capacity})(S/100); \qquad [A2]$$

$$\text{dissolved } O_2 = (PO_2)(O_2 \text{ solubility coefficient}); \qquad [A3]$$

$$\text{total } O_2 \text{ content of blood} = HbO_2 + \text{dissolved } O_2; \qquad [A4]$$

where HbO_2 is O_2 content of blood combined with Hb, and S is percent Hb saturation with O_2.

$$O_2 \text{ delivery rate} = (C_{aO_2})(\dot{Q}), \qquad [A5]$$

where C_{aO_2} is arterial O_2 content.

Dead space volume (V_D) [assuming equivalency of alveolar P_{CO_2} (P_{ACO_2}) and arterial P_{CO_2} (P_{aCO_2})] is

$$V_D = (V_T)([P_{aCO_2} - P_{ECO_2}]/P_{aCO_2}), \qquad [A6]$$

where V_T is tidal volume and P_{ECO_2} is expired P_{CO_2}.

Dead-space ventilation ($\dot{V}_D$) is

$$\dot{V}_D = (V_D)(f), \qquad [A7]$$

where f is respiratory rate.

Total minute ventilation ($\dot{V}_E$) is

$$\dot{V}_E = \dot{V}_A + \dot{V}_D, \qquad [A8]$$

where $\dot{V}_A$ is alveolar ventilation.

Alveolar ventilation is

$$\dot{V}_A = \dot{V}_E - \dot{V}_D. \qquad [A9]$$

The alveolar ventilation equation is

$$\dot{V}_A = (\dot{V}_{CO_2}/P_{ACO_2})(k), \qquad [A10]$$

where $\dot{V}_{CO_2}$ is rate of CO_2 production and k is a constant. Assuming constancy of $\dot{V}_{CO_2}$ and equivalency of P_{ACO_2} and P_{aCO_2} gives

$$\dot{V}_A P_{aCO_2} = k. \qquad [A11]$$

Alveolar P_{O_2} (P_{AO_2}) is

$$P_{AO_2} = (P_B - P_{H_2O})(F_{IO_2}) - (P_{ACO_2}/R). \qquad [A12]$$

(Note: For simplicity, a small correction factor normally added to the right side of the equation has been omitted.)

REFERENCES

1. West JB. Respiratory physiology—the essentials, 4th ed. Baltimore: Williams & Wilkins, 1990.

Books, Films, Tapes, & Software

Listing and Reviews of Books and Other Media
Note to publishers: Send review copies of books, films, tapes, and software to RESPIRATORY CARE, 11030 Ables Lane, Dallas TX 75229-4593.

Winning in Your Profession by Writing Books—A How-To Book for Professionals in the Biomedical and Behavioral Sciences and the Law, by Bowen Hosford. Hardcover, 196 pages, indexed. Springfield IL: Charles C Thomas Publishers, 1990. $36.75.

This book should be read by both the novice and the experienced author. If you have seriously thought about writing or editing a book but have not been able to make it happen, this monograph is a good "how-to." I have written or edited four books and find Hosford's advice to be right on target.

Hosford is a practicing attorney whose avocation is writing. His publications include a historical novel *The Grave of the Twin Hills* and two books on medical topics—*Making Your Medical Decisions: Your Rights and Harsh Choices Today* and *Bioethics Committees: The Health Care Providers Guide*. In preparation for this book, he taught an NIH graduate school course on book-writing.

He starts with a chapter entitled "Why Write Books?"—a frank discussion of what's in it for you as a professional, speaking to the self-discipline required. Chapter 2, "Fact vs Fiction," is interesting but adds little to what you probably already know. The third chapter provides some advice on how to select the type of book to write. The search for competing books is emphasized with suggestions for finding where new books would be welcomed by the profession. Part of this planning process includes identifying the target audience, and Hosford provides ideas for looking at the reading level of your peers.

Chapter 5 alone is worth the price of the book because it tells you how to find the time to write. Issues covered in this chapter include use of a home office, writer's block, writing with a computer, and other practical tips. What I like about this chapter is that the ideas flow from the experience of Hosford and several other seasoned authors whom he interviewed. His suggestions don't eliminate the work and dedication required, but they will save you time and increase your chances of bringing a book to print.

In Chapter 6, Hosford tells you how to prepare a book proposal and describes the elements that must be included. I find this to be one of the most important steps in writing a book because it requires you to write chapter outlines. The outlines and table of contents define the book so that the project can be sold to a publisher, and they provide the first blueprint of the work at hand. For potential authors who have never sold a book idea, this chapter is priority reading. Experienced authors will want to pull out their last proposal to see what they can add to the next book plan.

Most medical writers serve as their own literary agents and are faced with the publisher's "standard contract." Hosford does a good job in Chapter 7 of explaining why literary agents are usually not used by most medical writers. Better yet, in Chapter 8 he tells you how to sell a book idea without an agent.

Chapter 9 deals with the organization of the book and how to use formal outlines to guide the writing. His ideas on how to use a personal computer to replace file cards and his suggestions for organizing source material are very helpful. Comments about plagarism in this chapter are a sober reminder to all writers about the limits of using someone else's work. The author assumes that the reader has a graduate, medical, or law degree and already knows how to search the literature for source material.

Chapter 10 is titled "Writing a Book that People Will Read" and discusses many elements of good writing. A pitch is made for using the active voice, short sentences, and short words. But Hosford cautions against monotonous writing caused by adhering to rigid counts—that all sentences run under 20 words and most words be one-syllable ones. Other important topics included in this chapter are use of case studies and several grammatical tips: "Avoiding Adjectives and Adverbs," "Avoiding Preposition Pile-Up," "Tired Expressions," "Lulling (Boring) the Reader by Using Triplets," "Sexism in Pronouns," and "Using the First and Second Person." The importance of taking your writing through several revisions is also discussed in Chapter 10.

Chapter 11 deals with guarding against lawsuits and conflicts of interest. This chapter would be useful to authors who plan to use case studies in their books. Some important ideas about the importance of using sources from peer-reviewed periodicals are discussed in Chapter 11.

The last chapter has advice on how to boost your book's sales. Some interesting thoughts are presented such as promoting the book while writing it. The importance of feeding the publisher information about the potential readers of your book is emphasized, and Hosford discusses the professional groups that need to know about your writing project. Much of this chapter deals with promotional efforts beyond the needs of most medical writers. Two appendices are included. The first

lists books and periodicals directed at book writers and is a nice resource for further help with writing. The second appendix lists several macros for power users of personal computers. Unfortunately, the author covers only macros used on computers using a MS-DOS operating system. If you use another platform such as Apple, this appendix is worthless. Hosford should have included the same macro examples for Macintosh users.

In summary, if you are writing a respiratory care textbook or thinking about the project, read this book by Hosford. He does a good job of presenting his experiences from writing four books and those of several other authors. Seasoned book writers will also gain from reading what Hosford has to say about time management and other details of writing a book.

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Atlas of Bronchoscopy: Technique, Diagnosis, Differential Diagnosis, and Therapy, by Heinrich D Becker MD, Klaus Kayser MD, Volker Schulz MD, Siegfried Tuengerthal MD, and Hans-Hermann Vollhaber MD. Translated from German by Thomas J DeKornfeld MD and Heinrich D Becker MD. Hardcover, 182 pages, illustrated. Philadelphia: BC Decker, 1991. $125.00. Distributed by Mosby-Year Book, St Louis MO.

Conceptually, this atlas is divided into 6 sections, with the initial section briefly describing the indications and contraindications for bronchoscopy, rigid and fiberoptic endoscopes, anesthesia, monitoring, and obtaining specimens. The next section reviews the normal anatomy of the upper and lower airways, anatomic variations, and inflammatory disorders. This is followed by a section on lung parenchymal conditions that can be diagnosed via the airway, such as tuberculosis with microbiologic techniques and sarcoidosis with transbronchial biopsy. There is a detailed section on diagnosing neoplasia and following patients during and after therapy. The section on therapeutics reviews removal of secretions and foreign bodies, laser resection, endoluminal brachytherapy, fistula closure (fibrin glue), stent placement, and balloon tamponade. The final section reviews complications. A helpful index and 186 references (112 in English) are included.

The authors are members of the Pulmonary Disease Clinic in Heidelberg and represent internal medicine, pathology, and radiology. As expected, there are excellent color photographs of the airway printed on glossy paper. However, the authors add a new dimension with their multidisciplinary approach. They have provided pertinent photographs of the patient (at the bedside, in the operating room, through a thoracoscope), biopsy specimen (gross, microscopic: histopathology, cytopathology, immunopathology), radiographs (plain chest, CT scan, tomogram, angiogram), and special tests (flow-volume loop, pressure-volume curve, electron micrograph, and electron-absorption spectroscopy). Twenty-nine tables summarize information. The text succinctly describes the abnormalities shown, pathologic condition, diagnostic approach, therapy, and outcome.

The shortcomings are minor. For the most part, the figures are placed horizontally across the page, while the legends are listed vertically on the opposite page. It would be less distracting if the figures and legends appeared in corresponding positions as was done for asbestosis (Page 82) and metal dust lung (Page 83). Fibrinous tracheitis is called "fibrous" (Page 26, Figure 31) and the legend for Figure 152C is missing (Page 108).

By emulating an excellent case conference, the multidisciplinary approach establishes a new standard for teaching bronchoscopy. The authors are to be congratulated for their fine work. This atlas should be considered mandatory for bronchoscopists-in-training. Nurses and respiratory care practitioners who assist with bronchoscopy will also want to consult this book.

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Hemodynamics and Gas Exchange: A Clinical Guide, by Paul Ouellet RRT. Softcover, illustrated, 195 pages. Edmundston, New Brunswick, Canada: Marevie Inc, 1991. $32.95.

The author, Paul Ouellet, a respiratory therapist at the Edmundston Regional Hospital in New Brunswick, Canada, suggests that the purpose of this book is to merge the disciplines of gasometry (blood gases), CO-oximetry, and hemodynamics into an easy-to-use clinical reference. He has succeeded in combining these three separate topics into a concise clinical handbook.

At the very beginning of the book, the author presents detailed information about the conventions used to designate the variables of interest. In writing for "anyone involved in the treatment of critical care patients," Mr Ouellet has developed a text utilizing internationally accepted teminology that should be comfortable for most respiratory therapists to read.

In Chapters 2 and 3, gas exchange and hemodynamic elements are presented in a standard format. Each is reviewed in terms of symbol, definition, how measured, theoretical interest, reference values, and clinical in-

terpretation. I found this review helpful in enhancing my level of understanding before delving into more complex discussions concerning the physiologic interdependence of hemodynamics and gas exchange.

Chapters 4, 5, and 7 explain the relationship between hemodynamic and gas exchange elements in terms of data collection and physiologic interaction. Throughout the text, Mr Ouellet has utilized tables, figures, and graphs to effectively illustrate and clarify the discussions. For example, Figure 7-3, found on Page 120 (Chapter 7), displays a useful algorithm that summarizes the various mathmatical interactions that occur among the hemodynamic and gas exchange variables.

The basic principles of the pharmacologic control of preload, afterload, and contractility are illustrated in Chapter 6. Specific examples of the drugs used in the intensive care setting to manipulate the patient's cardiovascular status are also reviewed. An excellent table presented at the end of this chapter effectively summarizes the relationship between these cardiovascular variables and the therapeutic agents that were recommended to control them. This was achieved by displaying the coronary side affected, failing index, and recommended pharmacologic agent for each cardiovascular variable.

The occasional abbreviated presentation of information had me at times wishing for more in-depth discussion. This economy of explanation was noted several times throughout this clinical guide. For example, on Page 51, Mr Ouellet refers to the calculation of oxygen tension at 50% hemoglobin saturation using the Hill equation. Some of the symbols for this equation are not explained, and without further discussion the reader may be unable to calculate the presented variable. It should be noted, however, that this clinical guide is intended to be a concise reference for use in a clinical setting and, therefore, explanations are occasionally given with a minimal amount of discussion.

In general, the six appendices promise to be useful, outlining such procedures as radial artery line insertion and the use of a Swan-Ganz catheter. Other appendices list hemodynamic and gas exchange equations and a chart for estimating body-surface area. Appendix E, beginning on Page 172, is a somewhat redundant listing of all the gas exchange and hemodynamic parameters that were comprehensively listed in Chapters 2 and 3.

To summarize, this book is a concise clinical handbook that successfully explains the major concepts concerning hemodynamics and gas exchange. Although the book was written primarily for Canadian health professionals, respiratory therapists in the USA should have little difficulty understanding this text.

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Pediatric Home Care: Results of a National Evaluation of Programs for Ventilator-Assisted Children, by Lu Ann Aday PhD, Marlene J Aitken MAMS OTR/L, and Donna Hope Wegener MA. Illustrated, 464 pages, paperbound. Chicago: Pluribus Press, 1988. $34.95.

This book was written by Lu Ann Aday PhD, associate professor of behavioral sciences at the University of Texas School of Public Health; Marlene J Aitken, a doctoral student in the School of Public Health at the University of Illinois, and Donna Hope Wegener, a PhD candidate in the school of Social Service Administration at the University of Chicago. The book (designed to assess systems that provide care, train health personnel, and administer private and governmental agencies) contains more than 50 detailed and closely analyzed tables and figures, a methodologic appendix, and the complete caregiver questionnaire and key-informant questionnaire used to gather information on the programs.

The authors detail evaluation of three Special Projects of Regional and National Significance Ventilator-Assisted Care (SPRANS-VAC) programs, located in Louisiana, Illinois, and Maryland. Key points are reinforced and reiterated throughout the book. The book is easy to read and follow; each chapter begins with general information regarding data collection, and a summary of tables and contents is found at the end of each chapter. The authors' conclusions and evaluations are broken into 5 major categories: Program, Patients, Families, Policies, and Research.

The actual study emanated from the 1982 Surgeon General's Workshop on Children with Handicaps and their Families. The Division of Maternal and Child Health funded the SPRANS-VAC programs to develop regionalized, comprehensive, coordinated systems of care and a comprehensive evaluation of the implementation and impact of those programs. **Pediatric Home Care** is the result.

The authors assess not only the projects' effectiveness in meeting their grant objectives, but also their relevance for other communities and other types of chronically ill children.

Pediatric Home Care focuses on children with chronic physical illness, particularly those who require technologically sophisticated intervention. A conceptual model is introduced to explain the collection and analysis of data to evaluate the pro-

grams designed to create non-patient alternatives for one category of technology-assisted children.

Proponents of home care for chronically ill children not only predict reductions in cost but also improvements in the child's growth and development in the home. This study documents the effectiveness of these alternative interventions.

Chapter 1 reviews the literature current in 1988 and summarizes the study design and why each area was a focus for the program. Chapter 2 describes the methodology and analysis plan for the SPRANS-VAC evaluation. Chapters 3, 4, and 5 provide key-informant interview data to validate the descriptive profiles constructed from the administrative record materials and the principal data for describing the overall characteristics and implementation of the programs. Statistics on specific characteristics of the population and a description of the general socioeconomic and health-care system in the three program states were examined to provide an understanding of the environment into which the SPRANS-VAC programs were introduced. Chapter 4 presents the history of the programs, exemplifying ways in which different environments shape different program designs. Case studies describing the three programs are presented.

Chapter 5 develops performance profiles of the programs that reflect the implementation of the major program goals and draws implications from the program experiences in addressing these goals or, in some cases, electing to modify and/or deemphasize these goals over time. It was found in examining the programs that the goals and objectives (1) varied from program to program based on the environment, the participating staff, the target population, and of other factors; and (2) shifted within programs because of factors such as changing staff, environmental conditions, and resources.

Understanding the differences in the programs and the respective models that emerged in the three states is necessary to proceed to the evaluation of the ultimate impact of the programs on ventilator-assisted children and their families. Chapters 6-9 evaluate the ultimate impact of the SPRANS-VAC programs by looking at specific patient data. The authors considered quality of life of the ventilator-assisted children, impact on their families and principal caregivers, and, ultimately, cost of care at home compared to the hospital. At the time of publication, this information was the largest collected sample of ventilator-assisted children, their families, and the people who principally care for them. Both the families' and the programs' points of view are discussed.

In Chapter 9 the authors identify both direct and indirect costs of illness, institutional and non-institutional costs, distribution of costs by type of services, and relative costs in different states, concluding that the home care programs reduced the charges and costs of care for ventilator-assisted children. The bulk of their savings can be attributed to substitution of parental for professional nursing services in the home and to elimination of lab tests and procedures. Conclusion: Hospital *charges* exceed home costs by $793 per day on an average. Hospital *costs* exceed home costs by $418 per day on an average.

Chapter 10 addresses the implications of the findings for programs charged with responsibilities comparable to those of the projects reported here.

The authors conclude that the programs evaluated represent pioneer efforts to address many issues in forming a system of services for ventilator-assisted children. Strong interorganizational links and a stable program structure have been found to be the best predictors of success in developing a regionalized system of care and a comprehensive coordinated model of care. Financing of home care and financial problems experienced by families are the greatest predictors of family and caregiver stress once the child is home. The authors believe that health policymakers can address changes in both the mechanism and comprehensiveness of financing alternatives for chronically ill children.

I find the analysis of the three programs very accurate based on my personal involvement in one of the programs. This book can serve as a resource and reference guide for those parents, professionals, and policymakers involved in home care of technology-assisted children.

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Respiratory Infections in the Elderly, edited by Michael S Niederman MD. Hardcover, 320 pages, illustrated. New York: Raven Press, 1991. $65.00.

The elderly comprise one of the most rapidly growing segments of the American population and disproportionally account for a majority of hospitalized patients. Yet, most medical textbooks have been written by experts who practice in a university setting in which the patient population is often much younger. Therefore, a paucity of data have been reported specifically about the elderly. Realizing this, I had actually purchased this text prior to being asked to review it for the Journal. After reading and reviewing the text, I am very glad that I did make this purchase.

Dr Michael Niederman from State University of New York at Stony Brook has assembled a group of authors who are very well qualified and experienced in treating the elderly

pulmonary patient. The text is directed at physicians who treat elderly patients, although the experienced respiratory care practitioner will certainly be very interested in and will benefit from reading many, but perhaps not all, of the chapters. Two chapters that will be particularly interesting for respiratory care practitioners are Dr Niederman and Dr Alan Fain's chapter entitled "Community Acquired Pneumonia in the Elderly" and Dr Niederman's chapter on "Nosocomial Pneumonia in the Elderly," which are very well written and comprehensive, although they contain only a few illustrations. The chapter entitled "Slowly Resolving Pneumonia in the Elderly," also written by Niederman and Fain, is better illustrated. The authors are expert on this topic and have done a very fine job of summarizing multiple studies and in giving the reader a practical approach to this frequent clinical dilemma.

The chapters on "Epidemiology of Respiratory Infections in the Elderly," "Pulmonary Host Defenses in the Elderly," and "Pharmacology of Antimicrobials in the Elderly" are detailed reviews of their respective topics; however, they are of more interest to a physician or scientist than to respiratory care practitioners. I find the chapter on "Pharmacology of Antimicrobials in the Elderly" most helpful in sorting out the numerous antibiotics that have recently been developed and feel that this is a good overview. The chapters on "The Common Cold," "Influenza in the Elderly," and "Airways Infection" are also well written and may be of interest to the respiratory care practitioner. However, the chapter on "Airways Infection" is more superficial than the other chapters because it attempts to address several problems such as bronchiectasis and bronchitis without having the space to focus in completely on practical aspects. Dr William Stead, who has perhaps the most extensive experience with tuberculosis in the elderly, is co-author of the chapter on tuberculosis, a chapter that succinctly summarizes decades of data. Although there is only one chest radiograph, Tables 1 and 2 and Figures 1-4 are very well presented, and Table 3 and Figure 5 summarize treatment and outcome very well.

In conclusion, this book would serve as a good reference for the respiratory care practitioner. Again, I commend the authors for recognizing the need to direct more attention toward pulmonary disease in the elderly, and I hope that this will serve as a springboard for more focused research and writings on other aspects of pulmonary care in the elderly.

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ARKANSAS

ARKANSAS CHILDREN'S HOSPITAL

We wish to extend our appreciation to the dedicated and caring RESPIRATORY CARE PROFESSIONALS At Arkansas Children's Hospital.
(501) 320-3531

LIKE WHAT WE'RE DOING? TELL A FRIEND — CAN WE MAKE IT BETTER? TELL US!

LOUISIANA

LOUISIANA

OHIO

Building for the future

RESPIRATORY THERAPISTS

Tulane is a leading acute-care teaching and referral facility located in New Orleans. We are currently seeking highly motivated Registered Therapists for our Adult or Pediatric areas. LA licensure/eligibility required.

Our Respiratory Care Department offers the following:

- Supportive Medical Direction
- Participation in quality management
- Research and teaching opportunities
- Progressive Critical Care
- Physician rounds/care plan development
- State-of-the-art technology
- Adult and Pediatric Heart Transplants
- Neonatal Transport Team
- Code/ACLS/Intubation/Airway Management
- Arterial line insertions

Tulane provides an excellent compensation/benefits package along with relocation assistance and sign-on bonuses.

Qualified candidates submit resume to: Employment Supervisor, **TULANE UNIVERSITY MEDICAL CENTER HOSPITAL & CLINIC**, Human Resources Dept. No. SA30, 1415 Tulane Avenue, New Orleans, LA 70112. AA/EOE.

Tulane
UNIVERSITY MEDICAL CENTER HOSPITAL & CLINIC

AKRON, OH

RESPIRATORY CARE OPPORTUNITIES

Summa Health System, a 970-bed system dedicated to excellence in patient care, medical education and researeh, has the following positions available at Summa Health System.

- Supervisor, Respiratory Therapy 11pm to 7:30am shift, St. Thomas campus. Applicants must have a minimum two years respiratory experience and be a registered therapist.

- Part time evenings and night Registered Respiratory Therapists or certified Respiratory Technicians on both the Akron City and St. Thomas campuses.

Applicants must be eligible for Ohio license. Excellent benefits and competitive salary. Please submit resume or apply in person to: **Dept. of Human Resources, Akron City Hospital, 525 East Market St., Akron, Ohio 44309.** Equal Opportunity Employer.

SUMMA
Health System

Member Hospitals
Akron City Hospital
St. Thomas Medical Center

Alton Ochsner Medical Foundation Proudly Recognizes

NATIONAL RESPIRATORY CARE WEEK

The Alton Ochsner Medical Foundation Hospital salutes our staff of respiratory care professionals for their care and support.

These professionals are an integral component of the quality health care team at Ochsner.

Ochsner

LOUISIANA

Salutation for Respiratory Care Practitioners

For the Comfort and Support you give your patients. For the smiling reassurance you give worried parents. For working as a team when someone calls in sick. Or simply when everything falls apart.
For continuing your education and developing your skills. For the pride you show in your profession. For all that you are and yet to become. Congratulations! Have a great Day.

SCHUMPERT MEDICAL CENTER
We're here for Life!
A member of the Sisters of Charity of the Incarnate Word Health Care System

To: The Respiratory Care Staff
From: Your Fellow Staff Members at St. Ann's Hospital

Your hard work and dedication have produced a Respiratory Care Department that we are proud to have on our healthcare team. Thank you for the dedication that keeps St. Ann's Mission alive!

St. Ann's Hospital

St. Ann's Hospital
500 S. Cleveland Avenue
Westerville, Ohio 43081

MetroHealth Medical Center

Division of Pulmonary Services

CONGRATULATES

all their Respiratory Care Practitioners for their

- **Dedication**
- **Commitment**
- **High Standard of Care to all Patients**

INDIANA

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People talk. And the word is that the premier health care provider in the region also provides the number one career setting for over 2,000 employees. Why Memorial Health System? Our people tell us it's the opportunity to have a say in things, to continue learning at the forefront of medicine, to be recognized for quality, and to achieve lofty goals. There's much more to the picture. Be part of it all as:

• RESPIRATORY THERAPIST

As one of the largest, most progressive hospitals in Northwest Indiana, we maintain a dynamic environment, a strong sense of community, relocation assistance, and a highly competitive compensation/benefits package.

For more information about available positions call Carol Lyle-Ford, Allied Health Recruiter, **(219) 284-7407** or send resume to: Memorial Hospital of South Bend, 615 N. Michigan, South Bend, IN 46601. Equal opportunity employer.

Memorial
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PARK MEDICAL CENTER

GREAT OPPORTUNITIES AWAIT YOU AT THE PARK.

Backed by extensive resources and advanced capabilities, Park Medical Center is fast becoming recognized as one of the region's most progressive healthcare facilities. Today, we seek enthusiastic, goal-oriented professionals who are graduates from an AMA-approved school of respiratory therapy, with RRT or eligibility or CRTT status.

In addition to boasting the latest technology, The Park also offers a competitive salary and top benefits. For immediate consideration, please forward your resume to: Personnel Department, Park Medical Center, 1492 E. Broad St., Columbus, OH 43205. EOE m/f/d.

OHIO

OHIO

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JOIN THE TEAM-ORIENTED ENVIRONMENT OF ST. ANN'S HOSPITAL, A 180 BED COMMUNITY HOSPITAL LOCATED NORTH OF COLUMBUS, IN A BEAUTIFUL SUBURBAN SETTING. WE SEEK A REGISTERED RESPIRATORY CARE PROFESSIONAL TO MANAGE ALL FUNCTIONS INVOLVED WITH OUR BUSY PULMONARY DIAGNOSTICS LABORATORY. POSITION RESPONSIBILITIES WILL INCLUDE PULMONARY FUNCTION TESTING, BRONCHOSCOPY ASSISTANCE AND A.B.G. LAB.

ST. ANN'S HOSPITAL OFFERS A COMPETITIVE SALARY, COMPREHENSIVE BENEFITS AND A SUPPORTIVE WORK ENVIRONMENT.

INTERESTED CANDIDATES PLEASE SEND RESUME TO:

St. Ann's Hospital

HUMAN RESOURCES DEPARTMENT
500 S. CLEVELAND AVENUE
WESTERVILLE, OHIO 43081
EOE

Thanks !!!

To the Respiratory Therapy Section of the Pulmonary and Critical Care Medicine Department at the Cleveland Clinic Foundation.

Your dedication and efforts to provide the best possible respiratory care to the patients we serve is greatly appreciated!!

Keep up the great Work!!!

INDIANA

Respiratory Therapists

$1,000. Sign On Bonus

Respiratory Therapists
$1,000 Sign On Bonus

We are a highly motivated healthcare organization, seeking equally motivated & credentialed Respiratory Care Practitioners. Our rapidly expanding organization has what talented therapists are looking for:

- **Proven Track Record for Career Advancement Opportunities**
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Acmed

$1,000 Sign-On Bonus

CERTIFIED RESPIRATORY TECHNICIAN

Our hospital has full time positions for Respiratory Technicians on 2nd and 3rd shifts, including every other weekend. Salary based on experience, excellent benefit package plus $1,000 sign-on bonus.

Apply or send resume with cover letter to:
Human Resources Dept.

Jackson County Schneck Memorial Hospital
200 S. Walnut St.
Seymour, IN 47274
EOE

To: Riverview Hospital
Respiratory Care Practitioners

Congratulations for the recognition you well deserve during "Respiratory Care Week."

Thank You!!

From: Director of Respiratory Care and Administration
Riverview Hospital

KANSAS

RESPIRATORY CARE PRACTITIONERS

Asbury-Salina Regional Medical Center, 212-bed acute care regional referral medical center, is seeking qualified professionals to join our team. Minimum qualifications include RRT/CRTT or eligible.
Our Department, recently relocated into the medical center's $20 million expansion, performs PCIRV, surfactant replacement therapy and through our new "state-of-the-art" Pulmonary Physiology laboratory, metabolic nutritional studies.
Salina, a 1989 All-American City of 40,000+, is centrally located with clean air, excellent schools, and a large array of cultural, recreational and athletic activities and facilities. For more information or to apply, contact:

ASBURY-SALINA REGIONAL MEDICAL CENTER
PERSONNEL DEPARTMENT
400 SOUTH SANTA FE
SALINA, KS 67401
(913) 826-3154

KANSAS

RESPIRATORY THERAPISTS/TEAM LEADER
RESPIRATORY THERAPISTS

Positions now available at the University of Kansas Medical Center, a tertiary care hospital with 8 ICUs, excellent benefits, competitive salaries, and a comprehensive staff development program. New improved salary schedule. Assistance with NBRC exams and continuing education in Pediatric and Adult Critical Care Course. Kansas City is truly a "livable city" with the activities and opportunities of a metropolitan city, and the atmosphere of a friendly, small town. To apply call **913-588-3335** or send resume to Homer Rodriguez, Director of Respiratory Care, 5040 DELP Bldg., 39th and Rainbow Blvd., Kansas City, Kansas 66160-7109.

The University of Kansas Medical Center
AN EQUAL OPPORTUNITY AFFIRMATIVE ACTION EMPLOYER

Congratulations to the great Respiratory Care Staff at Lawrence Memorial Hospital, Lawrence, Kansas. Bret Casper, RRT was named "Employee of the Year". We're looking forward to another great year!

MICHIGAN

TO THE STAFF OF THE RESPIRATORY CARE DEPARTMENT DETROIT RIVERVIEW HOSPITAL

For your constant dedication to the highest standards of patient care and your outstanding commitment and dedication to the needs of the department; we wish to express our sincerest thanks and congratulations on a job well done.

Respiratory Care Management
Detroit Riverview Hospital

ILLINOIS

SOUTH CHICAGO COMMUNITY HOSPITAL

CONGRATULATIONS

To the RESPIRATORY CARE PRACTITIONERS at SOUTH CHICAGO COMMUNITY HOSPITAL, who meet the challenges of their career everyday with Dignity, Humor, and Grace.

From Respiratory Management and Administration at SCCH

EHS BRINGING THE BEST Together

SOUTH CHICAGO COMMUNITY HOSPITAL

GET THE
AARC ADVANTAGE
Call (214) 243-2272
For a Membership Application

JOIN THE FIESTA
at the AARC Annual Meeting
December 12-15
in San Antonio, TX

TEXAS

"After working in Respiratory Care at other hospitals, St. Luke's is like a breath of fresh air."
Mayo Mullone, BS, RRT, RCP
Respiratory Care

St. Luke's offers Respiratory Therapists like Mayo the opportunity to work more closely with doctors. As Mayo says, "Everybody on the team, including the doctors, operates on the same level with the same goal of helping the patient get better. I've been here five years; there's no better place to build a career than St. Luke's."

ST. LUKE'S
Episcopal Hospital

Step up to the benefits of working at St. Luke's in Houston's Texas Medical Center. Call collect (713)791-3995 or send your resume to our Employment Office (4-293), P.O. Box 20269, Houston, Texas 77225-0269.

Congratulations on Respiratory Week to "The Wean Team". Thanks for a job well done.

An Equal Opportunity Employer, M/F/H/V Non-smokers preferred

Critical Care Respiratory Therapists

Due to recent department restructuring, we now have opportunities for Registered Respiratory Therapists with Critical Care experience, (pediatric experience preferred).

Growth opportunities include specialization in ECMO and HFOV. Full time, 8-hour shifts, Monday through Friday (no weekends). For more information and appointment, please call the Allied Health Recruiter at 214/640-2163 or 1-800-852-0982.

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Equal Opportunity Employer M/F/D

To the staff of Respiratory Care Services,

I want to thank each and every one of you for your hard work, professionalism, perseverance, and support throughout the past year. Only through teamwork and trust in each others abilities have our efforts been successful. Respiratory Care Week is a time when we celebrate our profession and each other. **I want you and all of Respiratory Care to know how highly I think of you and how much you are appreciated.**

John D. Kimble Jr., RRT, RCP
Director - Respiratory Care Services

SCOTT & WHITE
2401 South 31st Street
Temple, TX 76508 EOE

For all your hard work and dedication,

We Thank You

Joan Goodpasture-Director-RRT
Dan Howard-Manager-RRT
Dan Nash-CRTT
Brenda Coen-CRTT
Donna Cooper-RCP
Shirley Summers-RCP

Harris Methodist Northwest

Respiratory Care Supervisors

Are you looking for an opportunity to exercise your managerial expertise in a teaching pediatric facility? Some attributes that set these positions apart from others are:

- No expectation to carry a patient load
- On-going in-house management development
- Involved with short and long term planning
- Management development of ICU team leaders and lead therapists in Acute Care
- Management of staffing and operations of the Emergency Referral Center, ICU, and Acute Care areas
- Non-salaried, with overtime available
- Up to $18.55 an hour (depending on experience)

Registered Therapist with management experience can contact the Allied Health Recruiter at 214/640-2163 or 1-800-852-0982 for further information and appointment.

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MONTANA

SUPERVISOR RESPIRATORY CARE

Graduate of 2 year school, Respiratory Care. Bachelor Degree in Health Care Administration, Business Administration, or Respiratory Therapy preferred. Registered Respiratory Therapist, 5 years clinical experience in advanced Respiratory Care, 2-3 years supervisory experience required.

For more information, call Human Resources:

(406) 444-2119

ST. PETERS
COMMUNITY HOSPITAL
2475 Broadway
Helena, MT 59601

The Administration & Respiratory Care Management Team
salutes
THE HIGH PLAINS BAPTIST HOSPITAL RESPIRATORY CARE TEAM
on a job well done!
High Plains Baptist Hospital
Amarillo, Texas
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NATIONAL RESPIRATORY CARE WEEK
October 4-10, 1992
RESPIRATORY CARE...CARES!

To our Respiratory Care Staff,

Thank You!

For your hard work, dedication, and commitment to patient care.

Saint Vincent Hospital and Health Center
Billings, Montana

CALIFORNIA

THANKS!

To all of our RESPIRATORY CARE PRACTITIONERS for their contributions as valuable health care team members at our hospital

SAN JOSE MEDICAL CENTER
SAN JOSE, CALIFORNIA

WASHINGTON

To Our Therapists Who Continually Live Up To Our Hospital Motto! Working Together To Make A Healthier "Community"
Community Memorial Hospital
Enumclaw Washington
Thanks You.

EARN CONTINUING RESPIRATORY CARE EDUCATION CREDIT WITH AARC VIDEOCONFERENCES

CALL (214) 830-0061

TEXAS

Respiratory Care Registered/Registry Eligible Night Shift (M-F)

The Respiratory Care Department is accepting applications for staff positions in its Acute Care areas. Responsibilities will include involvement in the Bone Marrow Unit, Cardiac Care, Liver Transplantation, ventilator management, and PRN rotation in the Critical Care and Emergency Referral Center areas. Lead therapists opportunities available:

- Up to $16.96 an hour (depending on experience and credentials)
- Wide variety of in-house CEU's offered
- No weekends
- Overtime options available
- Additional salary credits for pediatric experience.

For more information and appointment, please contact the Allied Health Recruiter at 214/640-2163 or 1-800-852-0982.

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Equal Opportunity Employer M/F/D

Learn What *Not To Do* With A COPD Patient

The Hospitalized COPD Patient: 10 Commandments for the Clinician — By David J. Pierson, MD. Takes you inside the decision-making process of caring for a respiratory care patient with chronic obstructive pulmonary disease. Details the "10 commandments" for the clinician to follow when encountering the 10 most serious problems the COPD patient can present to the hospital. Emphasis will focus on the *"what-not-to-do."*

Item VT29 — VHS (60 minutes)
$40 (Member $35)
Add $3 for shipping.

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or FAX Your Order to
(214) 484-2720

American Association for Respiratory Care
11030 Ables Lane
Dallas, TX
75229-4593

The Latest On

ARDS

ARDS Review: 1990 By Tony Dal Nogare, MD

ARDS was originally described in 1967 and has since undergone much scrutiny. This review of ARDS in 1990 discusses the latest developments in risk factors and treatment. Also covers the 5 diagnostic criteria that must be present to make an accurate diagnosis of ARDS, including clinical, radiographic, and physiologic criteria

Item VT31 — VHS (60 minutes)
$35 (Nonmember $40). Add $3 for shipping.

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American Association for Respiratory Care
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Work with your hospital public relations department to get the recognition you deserve. Keep them informed of your department's activities and personnel changes. If your PR department doesn't know about your recent promotion or your department's expansion of services, they certainly can't write an article about it for the hospital publication or local news.

PUBLIC RELATIONS BEGIN WITH YOU

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11030 Ables Lane
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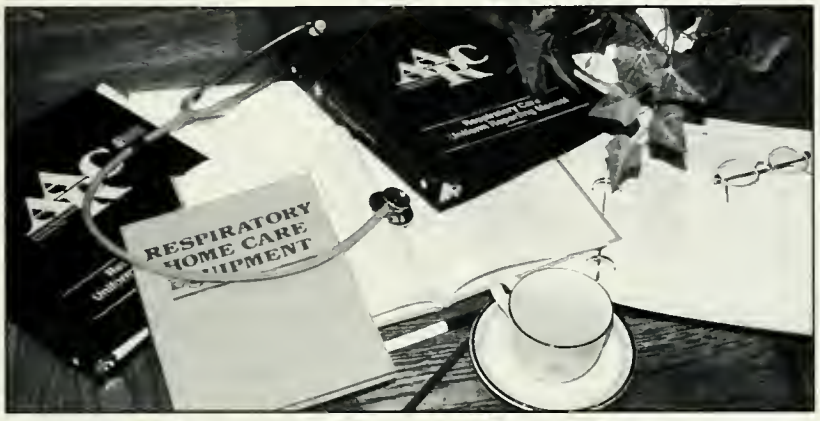
(214) 243-2272

A Commitment To Quality Health Care

RESPIRATORY CARE CARES



Put Today's Technology And Health Care Practices Into Action Efficiently With The Uniform Reporting Manual, 3rd Edition

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The Uniform Reporting Manual provides you with nationally recognized standards for documenting workload units and time standards.

- Patient Assessment Activities
- Bronchial Hygiene
- Supplemental Oxygen
- Airway Care
- Diagnostic Tests
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In addition, there are chapters on "Clinical Activities Without Time Standard" and "Management Support Activities." By the AARC. 1990, updated 1991. Binder. 123 pages.

The ONLY Single Source Of CGA, NFPA, And DOT Regulations

Respiratory Home Care Equipment exclusively covers home care equipment with practical applications and charts on oxygen concentrators, liquid units, air compressors, and home care ventilators. An invaluable book for the home care practitioner and equipment technician.

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- Description of home care devices.
- Cleaning, disinfecting, and monitoring procedures to minimize infection.
- Guides for educating staff on equipment, therapy, patient assessment, and safety.
- Procedures for gas administration and monitoring devices, humidifiers and nebulizers, artificial airways and resuscitators, respirators, and ventilators.

By Steven P. McPherson. Hardcover. 192 pages. 141 illustrations. 6 tables.

Researchers: If you only buy one book this year, buy Fundamentals of Respiratory Care Research

Once you have read *Fundamentals of Respiratory Care Research*, you will understand the practical importance of research to the field of respiratory care. And, you will possess the basic tools to carry out and critically evaluate your research efforts. Its step-by-step guidelines teach you implementation of a research protocol and the process of conducting a study. By Robert L. Chatburn, RRT, and Ken C. Craig, BA, RRT. 376 pages.

Item BK8

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RESPIRATORY CARE CARES

Maximize Your RC Knowledge With Independent Stu

Individual Independent Study Packages help you maximize your knowledge of respiratory care. Select from 31 different packages in Smoking Cessation, Clinical Science, Patient Evaluation, and Clinical Practice. Choose from the following list or order the entire set and save 10% off individual prices.

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Clinical Science

UPDATED! Bronchodilators: Sympathomimetic Amines

Understand the results of stimulating the autonomic nervous system and the use of sympathomimetic drugs to accomplish bronchodilation. Exposes you to basic aspects of adrenergic bronchodilators and the patient situations for which they are indicated.

Item CS14 — $10 Ea

UPDATED! Bronchodilators: Calculation and Preparation of Respiratory Medications

Teaches you how to calculate and prepare percentage solutions, ration solutions, solutions prepared from solutes less than 100 percent in strength, and how to prepare dosages from powdered drugs.

Item CS16 — $10 Ea

Classification of Mechanical Ventilators I

Outlines the basic concepts of ventilator classification and a mathematical model of the respiratory system that provides the basis for classifying ventilator control systems. Also teaches the specific criteria for determining whether a ventilator primarily controls pressure, volume, or flow.

Item CS18 — $10 Ea

Classification of Mechanical Ventilators II

You will learn detailed information about the control of ventilation in terms of switching from inspiration to expiration. You will also be introduced to the common drive mechanisms used in various mechanical devices.

Item CS19 — $10 Ea

Classification of Mechanical Ventilators III

Explores the output waveforms that ventilators produce and their clinical implications. Instructs in the various modes of ventilation in terms of the specific combinations of control characteristics along with ventilator performance testing and the practical application of ventilator classification.

Item CS20 — $10 Ea

Ventilation/Perfusion Relationships in Health and Disease

Covers the different degrees of hypoxia commonly encountered in patients with pulmonary disease. Focuses on ventilation/perfusion abnormalities, which account for much of the severe hypoxia in COPD, with alveolar hypoventilation also a contributing factor.

Item CS21 — $10 Ea

Transpulmonary Pressure Changes in Breathing

Discusses the pulmonary pressures and how they vary during normal breathing and in special situations. Instructs on barometric, alveolar, intrapleural, transpulmonary, chest wall, and transthoracic pulmonary pressures.

Item CS5 — $6 Ea

Resistance to Ventilation: A Second Look at Pulmonary Compliance and Resistance

Assists you in understanding the pulmonary characteristics of compliance and resistance, how these characteristics can change, and their influence on mechanical ventilator performance.

Item CS6 — $7 Ea

Practical Application of Gas Laws

A common-sense approach to the gas laws and how to apply them to respiratory care. Defines the laws of Boyle, Charles, Gay-Lussac, Dalton, Graham, and Henry.

Item CS7 — $8 Ea

Tonicity of Solutions and the Respiratory Tract

Gives you an understanding of how to predict the effects of therapeutic solutions on the respiratory tract and how to utilize them in the treatment of various respiratory tract pathologies.

Item CS8 — $6 Ea

Carbon Monoxide Inhalation: Introduction to Physiologic Effects and Respiratory Management

Explains the effects of carbon monoxide inhalation on oxygen transport mechanisms in the body and the use of CO in physiologic tests of pulmonary function. Also covers recognition signs and symptoms and treatment of CO poisoning and the equipment used to administer therapy.

Item CS9 — $7 Ea

Electrical Safety in Respiratory Therapy I: Basic Electrical Circuitry

Enables you to relate the basic principles of electrical theory to commonly used equipment and procedures in the clinical environment, and provides you with an understanding of electrical safety in respiratory care.

Item CS12 — $6 Ea

Electrical Safety in Respiratory Therapy II: Identification of Electrical Hazards

Helps you identify and, where possible, minimize or eliminate common electrical hazards. Teaches differentiation between electrical hazards and which patients and hospital personnel are exposed.

Item CS13 — $6 Ea

Microbiology for Respiratory Therapy: A Review of Microbial Growth and Cross-Contamination

Provides an overview of microbiology in respiratory care, including groups and characteristics of microbes, requirements for microbial growth, cross-contamination, and prevention of transmission.

Item CS17 — $9 Ea

Patient Evaluation

Pulmonary Function Assessment I: Basic Screening Studies

Understand simple spirometry results, the definition of obstructive and restrictive pulmonary disorders, and the basic pathology involved in each. Covers the four reasons for simple spirometry tests, the common parameters of lung function, and the results that are indicative of obstructive and restrictive pulmonary disorders.

Item PE3 — $8 Ea

Pulmonary Function Assessment II: Bedside Studies

Familiarizes you with the use and interpretation of bedside pulmonary function tests. Learn how pulmonary function test results are used to assess ventilatory ability and which tests are commonly used. Also, learn the measuring instrument required for each test, how it is performed, and what is significant about the results.

Item PE4 — $7 Ea

Pulmonary Function Assessment III: Lung Volume Determination and Closing Volume Studies

Assists in the recall of the eight subdivisions of total lung volume and their individual characteristics in health and disease. Discusses the measurement of lung volume by nitrogen washout, helium dilution, and closing volume techniques.

Item PE5 — $6 Ea

Body Plethysmography

Explains the theory of body plethysmography and its use in the measurement of FRC and airway resistance. Defines and describes Boyle's Law, thoracic volume, airway resistance and its normal values, and the measurement of airway resistance.

Item PE6 — $9 Ea

Sources of Error in the Determination of Blood Gas Values and pH

Familiarizes you with the errors that may occur in the analysis of blood gases and pH. These are often the most important laboratory data used in the diagnosis and treatment of pulmonary disease, and errors in these values can result in deleterious effects on patient care.

Item PE7 — $10 Ea

Temperature Adjustment of Blood Gases and pH

Teaches you the effects of abnormal body temperature on blood gas and pH values. Blood gas values and pH are determined at 37 degrees Celsius, and this package teaches you how to adjust these values.

Item PE8 — $10 Ea

Measurement of Static Compliances and Dynamic Characteristics Curves During Mechanical Ventilation

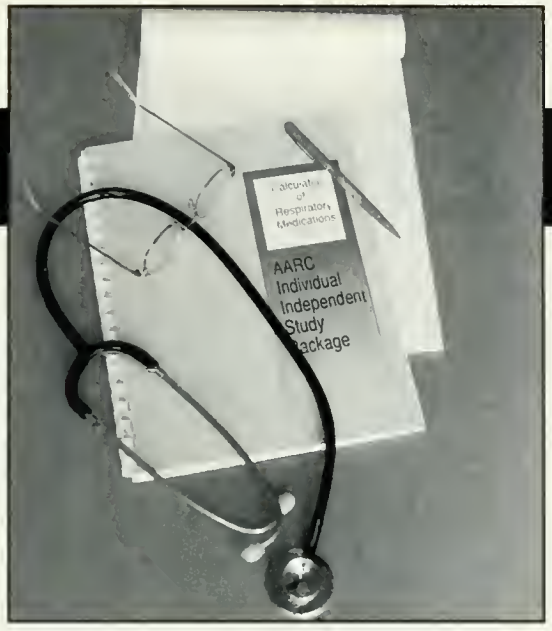
Learn how to compute and record static compliance curves and dynamic characteristics. And, learn to describe the procedure of obtaining pressure-volume measurements and interpret the compliance characteristics measurements.

Item PE9 — $7 Ea

Arterial Blood Gas Interpretation

Teaches you ABG interpretation in order that the prescribed therapy can be administered in a knowledgeable manner. Describes a systematic method that allows you to correctly classify the acid/base dysfunction and to relate the diagnosis concisely and coherently.

Item PE10 — $10 Ea



Clinical Practice

UPDATED! Chest Tubes and Pleural Drainage

Helps you understand the purpose of pleural drainage, how it might affect the patient's respiratory status, and what precautions you must take when working with patients who are receiving pleural drainage.

Item CP3 — $10 Ea

Tracheal Intubation I: Upper Airway Anatomy and Goals of Intubation

After completing this ISP, you will understand the rationale for tracheal intubation and be able to identify the important landmarks of upper airway anatomy.

Item CP4 — $6 Ea

Tracheal Intubation II: Routes of Intubation

Describes the four routes of tracheal intubations and some advantages and hazards of each. Also presents the process for selecting the most suitable route in a given situation.

Item CP5 — $6 Ea

Tracheal Intubation III: Equipment Procedures for Intubation

Covers the selection of the proper equipment necessary to perform endotracheal intubation and to ensure that they are in working order.

Item CP6 — $8 Ea

Respiratory Management of Neuromuscular Crisis

Teaches respiratory management of patients with ventilatory failure caused by a neuromuscular disorder. Provides a basic understanding of how neuromuscular conditions lead to ventilatory insufficiency and the considerations to be taken when working with these patients.

Item CP7 — $7 Ea

UPDATED! Respiratory Management of Flail Chest

This study package helps you increase your understanding of the pathophysiology of flail chest and the respiratory management of patients who have sustained chest wall trauma that results in a flail chest.

Item CP8 — $10 Ea

Respiratory Management of Head Trauma

Teaches identification of the five physical signs indicative of head trauma and explains the development of respiratory failure secondary to trauma. Also discusses airway management, drug therapy, ventilator parameters, acid-base status, and measures to be taken to maintain the appropriate pH, P_{aCO_2} and P_{aO_2}.

Item CP9 — $7 Ea

RESPIRATORY CARE CARES

Smoking Cessation

Nicotine Dependency Evaluation and Treatment

Helps you understand the physiologic effects of nicotine and the tests and questions used to evaluate dependency. Provides an understanding of nicotine, its effects on brain chemistry, its measurement in bodily fluids, and value of self-tests to determine addiction level. Also teaches about nicotine replacement and how to enhance it with behavioral counseling.

Item SC1 — $10 Ea

Bedside Counseling of the Hospitalized Smoker

Prepares you for the role of a smoking cessation counselor to hospital inpatients by teaching assessment and bedside counseling. Help patients cope with nicotine withdrawal, increase motivation for permanent cessation, and understand the factors contributing to relapse.

Item SC2 — $10 Ea

RESPIRATORY CARE CARES

Be On The Cutting Edge of Today's Respiratory

Convenient one-hour videotapes put you on the cutting edge of respiratory care with the newest technologies and techniques. View and review them at your leisure... perfect for staff review or at-home study.

Price: $40 Ea

(Member $35)

Available only in VHS

Complete Set!

Item VT88

$760

Set of 20

(Member $665)

Hospital Acquired Pneumonia

By Galen B. Toews, MD. Provides information on the epidemiology, pathogenesis, and management of nosocomial pulmonary infections. **Item VT36**

Smoking Cessation: Intervention Techniques For the Respiratory Care Practitioner

By Kathleen A. Smalky, MD, MPH. Covers four major aspects of smoking cessation — the impact of smoking on illness and mortality, behavioral components, current cessation programs, and effective intervention. Discusses identification of the physically addicted smoker and the effects of nicotine withdrawal. **Item VT35**

Nutrition and Respiratory Care

By Rich Branson, RRT. Explains the relationships and interactions of malnutrition on ventilatory drive, respiratory muscles, lung structure, and immunity. Further discusses the effects of nutrients on the respiratory system, particularly the nutritional support needed by mechanically ventilated and COPD patients. **Item VT34**

Uses and Abuses of Non-Invasive Monitors in Respiratory Care

By Dean Hess, RRT. Presents a discussion of how much noninvasive monitoring is needed, proof of false positives, its financial impact, and its future. Discusses the various methods: transcutaneous, pulse oximetry, and capnography. **Item VT33**

Sleep Disorders

By Brian Foresman, MD. A discussion of the physiology of sleep and the kinds of respiratory and nonrespiratory sleep disorders seen in the hospital. Discusses how to spot sleep apnea, the problems caused by the inpatient hospital setting, sleep disorder diagnosis, and treatment. **Item VT32**

Sleep Apnea

By Alan K. Pierce, MD. Explains how sleep stages are related to respiratory patterns and blood gas values in both normal and abnormal subjects. Includes a discussion of the criteria for defining the sleep apnea syndrome and the distinguishing features of central, obstructive, and mixed causes of apnea. Also addressed is the efficacy of medical treatment to correct specific types of sleep apnea. **Item VT11**

Monitoring Respiratory Mechanics During Mechanical Ventilation

By Robert L. Chatburn, RRT. Explains how physical and mathematical models are developed and applied and how they are incorporated into ventilator design to provide estimates of mechanics and some of the problems that can develop because of the limitations of the models. **Item VT30**

Pressure Support Update

By Neil MacIntyre, MD. Reviews pressure support ventilation and how it is used. Explains waveforms for airway pressure, flow, and volume. Also illustrates the measurement of inspiratory muscle loads by pressure-time index and muscle $\dot{V}_{O_2}$, and demonstrates how muscle efficiency changes as the character of work changes. Includes examples of low- and high-level PSV for elimination of the imposed work of breathing and as a weaning tool. **Item VT28**

Managing the Work of Breathing During Mechanical Ventilation

By John Marini, MD. Learn about the work of breathing and ways in which it can be minimized in the clinical setting on patients receiving mechanical ventilation. Focuses on diminishing the breathing workload through quantifying the effort during mechanical ventilation via direct measures such as oxygen consumption, electromyography, pressure-time index, and external work of breathing, as well as indirect measures. **Item VT27**

Modes of Conventional Ventilation

By Robert M. Kacmarek, PhD, RRT. A review of mechanical ventilation is presented, including the rationale for movement from one generation of ventilators to another. Ventilator modes discussed include control, assist/control, intermittent mandatory ventilation, synchronized intermittent mandatory ventilation, mandatory minute ventilation, and pressure support ventilation. **Item VT19**

Pressure Support Ventilation

By Neil MacIntyre, MD. This presentation defines and describes the physiological considerations attributed to PSV and the clinical situations when PSV may be useful. Includes comparisons when low-pressure levels and high-pressure levels of PSV are offered. Discussion includes using PSV to help overcome resistance for intubated patients and as an augmented ventilatory mode of weaning. **Item VT17**

Theory and Application of Neonatal Ventilation

By Robert Chatburn, RRT. Knob-turning in the neonatal intensive care unit should be a profound activity because it often has profound consequences. Adjustment of a single control on a ventilator generally has multiple effects, and thorough consideration of how controls are interrelated is essential for optimum care. Presents a well-organized and systemic approach for managing mechanical ventilation. **Item VT25**

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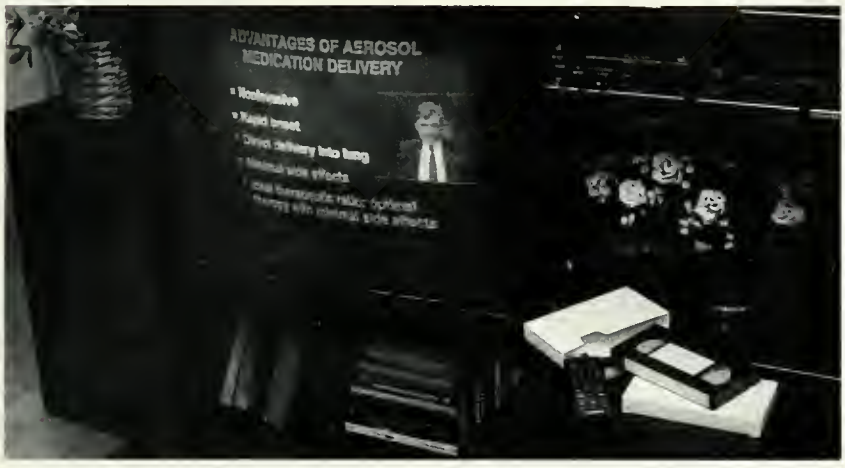

Fetal Lung Development

By Charles Rosenfeld, MD. Examines the four anatomical phases of fetal lung development along with a discussion of the biochemical development of the fetal lung through gestation. Includes a description of the substances in surfactant, the importance of their timely development, and the methods used to assess fetus survivability by using tracheal aspirant to identify the necessary ratios of the phospholipids making up surfactant. **Item VT23**

ARDS Review

By Tony Dal Nogare, MD. Discusses the latest developments in risk factors and treatment. Also covers the five diagnostic criteria that must be present to make an accurate diagnosis of ARDS, including clinical, radiographic, and physiologic criteria. **Item VT31**

Practical Management of ARDS

By David J. Pierson, MD. Adult respiratory distress syndrome is defined in this informative tape and its clinical features described, including incidence of risk factors and clinical predictors. Also extensively discussed is the use of PEEP to treat ARDS, including goals, complications, best or optimal PEEP levels, PEEP trials, and PEEP withdrawal, as well as general treatment, prognosis, and sequelae of ARDS. **Item VT16**

Tissue Oxygen Delivery

By David R. Dantzker, MD. Discusses the relationship between adequate tissue oxygenation and adequate energy production, the development of lactic acidosis, and the probable role of tissue hypoxia in the multiple-organ failure of ARDS. Also discusses the factors that govern oxygen transport to the tissues and the variables that reflect the adequacy of tissue oxygen transport. **Item VT26**

The Hospitalized COPD Patient: 10 Commandments For the Clinician

By David J. Pierson, MD. Takes you inside the decision-making process of caring for a respiratory care patient with chronic obstructive pulmonary disease. Details the ten rules for the clinician to follow when the COPD patient presents to the hospital. Emphasis is on the "what not to do." **Item VT29**

Pulmonary Rehabilitation

By John E. Hodgkin, MD. Provides an overview of the sequence for pulmonary rehabilitation. Learn candidate evaluation and selection, rehabilitation team establishment, identification of short- and long-term goals, program components, assessment of patients' progress, and long-term follow-up. Discusses aerosol therapy, IPPB, oxygen therapy, and chest physiotherapy in the treatment of COPD. **Item VT7**

Clinical Use of the Swan-Ganz Catheter

By John Marini, MD. Clinical applications of data obtained by Swan-Ganz catheter placement and the situations in which SGC placement are useful are described in this video. The clinical value of the clinical variables monitored by SGC and the complications of its placement are detailed, including "damping" of the waveform, "overwedging," and optimal lung zone placement. **Item VT22**

Drainage of the Pleural Space: Management of Chest Tubes and Bronchopleural Air Leak

By Martha L. Tyler, RN, RRT. Learn the physiologic effects of abnormal pleural space function and the potential problems associated with chest tubes stripping, difficulties of bronchopleural air leaks with mechanical ventilation, the therapeutic goals of chest tube placement, and techniques for maintaining gas exchange with air leaks. **Item VT21**

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Letters

Objections to Postural Drainage Therapy Guideline

I have read with interest the "AARC Clinical Practice Guideline: Postural Drainage Therapy."[1] I am a physical therapist who has practiced pulmonary, or chest, physical therapy for more than 20 years. I found it most unusual that in your first section of the article, PDT 1.0 Procedure, you have equated the term "Postural Drainage Therapy" with chest physiotherapy, chest physical therapy, etc, and referenced papers 7-12; none of which were written by pioneers in this area of care. It seems most unusual to me that the authors have not gone to the very sources of these techniques, which stem from as early as the 1900s and move on forward with definition of chest physical therapy and the various techniques that have resulted or been used in this area of care.

I have numerous variations in perspective throughout this article, but I am most concerned with PDT 5.0 Contraindications. Under 5.0 Positioning, you have listed that all positions are contraindicated for rib fracture with or without flail chest (5.1.1.12). In my clinical experience, I have found that no patient requires more care in terms of clearing excess secretions or preventing or reversing atelectasis than the individual with a rib fracture. As well, I have found patients with rib fractures, including those who have iatrogenically induced fractures due to a thoracotomy, respond very well to position changes. In fact, lack of position change will promote or induce greater atelectasis or hypoventilation. I urge you to reconsider. I can think of nothing more harmful for a patient who is hypoventilating due to a rib fracture than to be kept in a single position. Even more curious is that the authors have very clearly delineated that all positions are contraindicated. I find it very interesting that the authors have used the term "all positions are contraindicated for" and then have a large list. What position is the patient to be in if all positions are contraindicated? This seems to be incongruous and I am very unclear as to why this terminology was used.

As well, I cannot imagine that a person with a surgical wound or healing tissue, 5.1.1.1.3, is not allowed to be turned in any manner (ie, that all positions are contraindicated). I could go through the list for each item here because according to what is printed, all these patients should not even be rolled, and I doubt that that is really what the authors wanted to say.

Under 5.2, External Manipulation of the Chest, once again it is unclear to me what your guidelines are offering. The guidelines appear to be incorrect or not accurate as printed. For example, the person with a recent skin graft may have large amounts of retained secretions and be able to tolerate vibration at a site distal to the skin graft. The skin graft itself would not contraindicate the vibration, but vibration may be contraindicated over the skin graft. The same applies for anyone with a burn. In our clinical experience, we have used vibration and percussion on patients with lung contusion (5.2.7) and have found that manual techniques can greatly facilitate clearing the retained blood clots that can result from the lung contusion. The manual techniques do not hinder the healing of the individual, but by facilitating clearance of the blood clots, the patient can show a dramatic shift with increased oxygenation and change in x-ray. Once again I would ask that this list be re-examined and guidance be offered in a more specific manner so that the clinician reading it understands the parameters of decision making that will facilitate care to the patient who has any of these diagnoses.

I would also like to comment on PDT 6.0, Hazards/Complications, 6.2 Increased Intracranial Pressure. Mackenzie et al[2,3] found that the intracranial pressure increase was in fact more due to position change than to the chest physical therapy technique. Once the person has been rolled, the intracranial pressure returned to normal. This happened whether the person was simply rolled side to side or in fact was placed in a more active or aggressive postural drainage position. I do not believe that 6.2 addresses this variation that can occur due to simple repositioning. The outline does not allow the clinician to understand how to differentiate a transient change in intracranial pressure from simple movement versus an increased intracranial pressure due to a position that is not appropriate.

I would also like to comment on PDT 9.0, Assessment of Outcome, 9.1, Change in Sputum Production. It has been our clinical experience as well as that of others (Mackenzie et al[3]) that small amounts of secretions cleared from the acutely ill patient who is mechanically ventilated may in fact produce very dramatic changes in terms of resolution of atelectasis on x-ray, increased P_{aO_2}, and dramatic changes in compliance. Consequently, I do not agree with the statements that a sputum production of less than 25 mL a day means that treatment is not indicated in the acutely ill, mechanically ventilated patient. I believe that this is a gross oversight in terms of who and how this treatment should be delivered and is not adequately addressed in this particular guideline.

The authors comment that with effective therapy, breath sounds may worsen (PDT 9.0, PDT 9.2). How-

ever, breath sounds may also dramatically improve (ie, with adequate treatment, formerly decreased or absent breath sounds may become audible in a particular lobe). I do not believe that this is a worsening of breath sounds, but in fact a correction or optimization of breath sounds, and I do not agree with the wording.

Thank you for the opportunity to comment on this article.

Colleen M Kigin MS MPA PT
Director
Physical Therapy Services
Massachusetts General Hospital

Associate in Anesthesia
Harvard Medical School
Boston, Massachusetts

REFERENCES

1. American Association for Respiratory Care, Working Group. AARC Clinical Practice Guidelines: Postural drainage therapy. Respir Care 1991;36:1418-1426.
2. Mackenzie CF, ed. Chest physiotherapy in the intensive care unit, 2nd ed. Baltimore: Williams & Wilkins, 1989.
3. Mackenzie CF, et al. Changes in total lung/thoracic compliance following chest physiotherapy. Anesth Analg 1980;59(3):207-210.

Dean Hess and Pat Brougher respond:

We thank Ms Kigin for her thorough and thought-provoking response to the AARC Clinical Practice Guideline (CPG) on Postural Drainage Therapy. Before responding to the specific points raised by Ms Kigin, we would like to clarify several points about the CPG.

Considerable thought and debate goes on in the Working Groups as each CPG is formulated. Further, the Working Groups consist of persons with a broad background and experience. In the case of the Postural Drainage Therapy CPG, the Working Group included persons with 20 years or more of respiratory care experience. The Working Group also included a critical care physician and a physical therapist.

The Postural Drainage CPG was mailed to about 650 reviewers, and many of these provided comments before publication.

Guidelines are just that—*guidelines*. They are not rigid rules. They are designed to allow the sophisticated practitioner to make informed decisions.

As much as possible, the CPGs are based upon the peer-reviewed literature. Anecdotal evidence (ie, "clinical experience") cannot be independently confirmed, and is either accepted or rejected based upon the reputation of the author.

Regarding the specific concerns of Ms Kigin, we would offer the following.

The original papers of the pioneers in chest physical therapy are not provided in the reference list of the CPG because they were not believed to be relevant. The purpose of the Guideline was not to review the history of the subject. The authors of this CPG were perhaps bold in suggesting a new name for this therapy. Interestingly, however, at least one editorial[1] and one review[2] published since the CPG have suggested that renaming might be appropriate.

With few exceptions, the contraindications for Postural Drainage Therapy are relative (as stated in the Guideline). Perhaps the most important part of the Contraindications section of the CPG is not the list of contraindications, but rather the opening sentence of the section: "The decision to use postural drainage therapy requires assessment of potential benefits versus potential risks." For the example of a trauma patient with rib fractures who has retained secretions and lobar atelectasis, a very cogent case can be made that the potential benefits outweigh the risks.

It might actually be more reasonable to focus on indications rather than contraindications. In many hospitals, we suspect that there are many more patients who receive unnecessary postural drainage therapy (ie, no indications present) than there are patients who are denied therapy (ie, contraindications present).

The benefits of turning are important, and many patients require no pulmonary therapy other than frequent position changes. Frequent position changes, which should be part of traditional nursing practice, are different from postural drainage, which involves specific positioning to drain a specific lung segment (and typically requires the expertise of a respiratory care practitioner or a physical therapist).

Regarding the contraindications to external manipulation of the thorax—again all of these are relative, not absolute. Using Ms Kigin's example, all (other than perhaps the most unsophisticated reader) will recognize that this means that vibration and percussion are contraindicated over the site of the graft. Again, the intent of a guideline is not to be a rigid unbending rule.

Certainly many patients with head injury can safely tolerate postural drainage therapy. However, it is prudent to recogize that some patients with severe head injury and intracranial hypertension tolerate absolutely no change in body position. It is irresponsible not to recognize this. The determination of an increase in intracranial pressure that is not appropriate is a medical decision made best by the neurosurgeon or trauma surgeon. We know trauma surgeons who write postural drainage therapy orders with specific instructions relative to tolerable intracranial pressure increases during therapy. Some patients may require specific therapy to control intracranial pressure during treatments

(eg, mannitol, hyperventilation). However, the management of intracranial pressure is beyond the scope of this CPG.

The authors of this CPG agree that postural drainage therapy may be indicated in mechanically ventilated patients who produce less than 25 mL/day of sputum. This is adequately covered in the introduction to "PDT 9.0 Assessment of Outcome:" "Not all criteria are required to justify continuation of therapy (eg, a ventilated patient may not have sputum production > 30 mL/day, but have improvement in breath sounds, chest x-ray, or increased compliance or decreased resistance)."

The intent of the CPG relative to breath sounds (PDT 9.2) was to point out that loosening of secretions may result in worsening of breath sounds. Clearing of those secretions (eg, cough, suction) often results in clearing of breath sounds (as stated in the Guideline). The point is that loosening of secretions without their removal may result in worsening of breath sounds, whereas loosening of secretions and their removal results in improvement of breath sounds.

Again, we appreciate the comments of Ms Kigin. However, we suggest that readers of this and other CPGs view them in their entirety, and not as isolated statements within a Guideline.

Dean Hess MEd RRT
Chairman, Clinical Practice
Guidelines Steering Committee

Assistant Director
Department of Research
York Hospital
York, Pennsylvania

Pat Brougher BA RRT
Clinical Practice Guidelines
Project Manager

Editor, RESPIRATORY CARE
Dallas, Texas

REFERENCES

1. Lewis RM. Chest physical therapy: time for redefinition and renaming (editorial). Respir Care 1992;37: 419-421.
2. Dean E, Ross J. Discordance between cardiopulmonary physiology and physical therapy: toward a rational basis for practice. Chest 1992;101:1694-1698.

Bug? Yes. Insect? No.

In response to the letter describing a 'foreign body' in a ventilator circuit[1]—a spider is not an insect.

David J Pierson MD
Medical Director
Respiratory Care
Harborview Medical Center
Professor of Medicine
University of Washington
Seattle, Washington

REFERENCES

1. Theroux MC, Zagnoev M, Ponce C. Could you be the reason for a "spider in the airway?" (letter). Respir Care 1992;37:950.

The editor responds:

My oversight, Dr Pierson! Allow me to quote an appropriate (?) bit from Shakespeare's *A Midsummer Night's Dream.*

> Weaving spiders come not here;
> Hence, you long-legg'd spinners, hence!
> Beetles black, approach not near;
> Worm nor snail, do no offence.

Pat Brougher BA RRT
Editor, RESPIRATORY CARE
Dallas, Texas

Baggers, Not Beggars

AAAARGGHH!! In my intubation letter published in August,[1] a typo changed "Silent, obedient baggers" to "beggars."

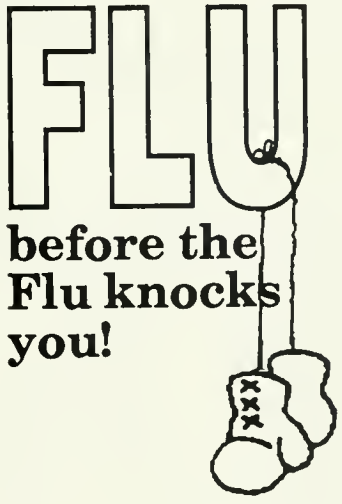
Ah, well, I guess baggers can't be choosers.

Tom Burns RRT
Phoenix, Arizona

REFERENCES

1. Burns T. Emergency care and ACLS (letter). Respir Care 1992;37:947-948.

Be a fighter. Knock out the



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For your information, answers to the 50 questions for ***CRCE through the Journal***, which appeared in the July issue of RESPIRATORY CARE, are given below. No scores will be available from the AARC until 1992 transcripts are released in early 1993. Deadline for submission of answer sheets for CRCE credit was August 15, 1992.

The correct answers to the questions are shown below.

1. a	14. b	27. e	40. c
2. e	15. b	28. b	41. e
3. e	16. e	29. b	42. d
4. b	17. e	30. e	43. e
5. c	18. e	31. c	44. b
6. e	19. e	32. b	45. c
7. b	20. e	33. b	46. a
8. a	21. b	34. e	47. e
9. e	22. e	35. e	48. d
10. e	23. e	36. b	49. d
11. e	24. d	37. e	50. b
12. d	25. b	38. a	
13. e	26. a	39. e	

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Three awards of $333 each are to be awarded to the authors of the three best features from Test Your Radiologic Skill, Blood Gas Corner, and PFT Corner accepted for publication from November 1991 through October 1992. All three (or none) of the features may be chosen from a specific category (eg, all three may be chosen from Blood Gas Corner).

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AARC & AFFILIATES

October 14 in Auckland, New Zealand. The NZSRC presents its Annual Scientific Meeting at the Aotea Centre. The meeting precedes the Australian-New Zealand Intensive Care Society Conference. Contact Graeme A'Court, PO Box 10148, Balmoral, Auckland, New Zealand. (643) 640640.

October 16 in Long Island, New York. The Southeastern Chapter of the NYSSRC presents its 24th Annual Symposium, "Focus on Technology," at the Marriott Hotel in Uniondale. Speakers include Michael McPeck, Nathan Seriff, Joseph Lore, Steven Marzo, Margaret McGovern, and AARC President Bob Demers, who present "Clinical Hazards Associated with Aerosols." Contact Ken Nugent RRT (516) 444-8511.

October 28-29 in Sturbridge, Massachusetts. The MSRC presents its 15th Annual Meeting and Exhibition at the Sturbridge Host Hotel and Conference Center. Topics include adult and neonatal ventilator management, the RCP's role in smoking cessation, and interpretation of ventilator graphics. Events include Sputum Bowl, golf tournament, and awards banquet. Contact Bill McGarry CPFT RRT, Respiratory Care, Beth Israel Hospital, 330 Brookline Ave, Boston MA 02215. (617) 284-0782.

October 30 in Bear Mountain, New York. The Hudson Valley Chapter of the NYSSRC presents its Annual Educational Seminar, "Challenging Issues in the Respiratory Care Environment," at the Bear Mountain Inn. Topics include patient assessment, infection control, and risk management. Contact Larry Lyman (914) 358-6200, pager 600.

November 17 Nationwide. The AARC, in conjunction with VHA Satellite Network, presents "Prevention of Postoperative Atelectasis and Pneumonia," one in a series of live satellite videoconferences titled "Professor's Rounds in Respiratory Care." Featured presenters are Dr David J Pierson and Richard Branson RRT. Site registration for entire staff is $245 for AARC members. Call (214) 830-0061.

February 16-19 in Reno, Nevada. The American Lung Association of Nevada and The Nevada Society for Respiratory Care present the 12th Annual High Sierra Critical Care Conference at the Peppermill Hotel Casino. Topics include adult, pediatric, and neonatal critical care issues. Contact Donna Turner, American Lung Association of Nevada, PO Box 7056, Reno NV 89510. (702) 829-5864 (9 AM-3 PM PST).

OTHER MEETINGS

October 11-13 in Arlington, Virginia. The first National Asthma Conference is scheduled to include a workshop specifically for respiratory care practitioners. The workshop has been approved for 9 continuing education credits by the AARC, and is open to any respiratory care practitioner who wishes to attend. Contact NHLBI (301) 951-3275.

October 11-18 in the Caribbean. LIFE Unlimited presents a "Western Caribbean Cruise" for pulmonary patients, their families, and friends aboard the SS Seabreeze. Cruise from Miami to Grand Cayman, Montego Bay, and Cozumel with private escorted tours and 24-hour professional services. Reservation deadline is Sept 4. Contact Dave Robbins, LIFE Unlimited, 17101 SW 200 St, Box Z28, Miami FL 33187. Call (800) 621-4571 or (305) 441-6819.

October 21-23 in Salt Lake City, Utah. The Centers for Disease Control, the Association of State and Territorial Health Officials, and the Association of State and Territorial Chronic Disease Program Directors cosponsor the 7th National Conference on Chronic Disease Prevention and Control, "Prevention: Can It Be Marketed, Managed, and Make a Difference?" at the Salt Lake Hilton. Contact Jack Friel, Chief, Conference Management Operations, National Center for Chronic Disease Prevention and Health Promotion, CDC, 1600 Clifton Rd NE, Mailstop K-43, Atlanta GA 30333. (404) 488-5390.

October 22-23 in Birmingham, Alabama. The Children's Hospital of Alabama, Department of Respiratory Care Services, hosts its 2nd Annual Perinatal/Pediatric Respiratory Care Clini-Course at the Birmingham-

Jefferson Civic Center. Topics to be discussed by guest lecturers include fundamentals of respiratory care research, efficacy of CPT in neonates, new approaches to the treatment of cystic fibrosis, and optimal delivery systems for continuous aerosolized medications on and off mechanical ventilation. Contact Susan Forrest (205) 939-9675.

October 23 in Dallas, Texas. Baylor University Medical Center hosts "Hand In Hand: Team Approach to Caring for the Pulmonary Patient" at Cityplace Conference Center. The seminar is designed for nurses and respiratory care practitioners caring for pulmonary patients in critical or acute care settings. Contact Barbara Grayson, Baylor University Medical Center, Continuing Education, 3500 Gaston Ave, Dallas TX 75246. (214) 820-2317.

November 18-21 in Miami Beach, Florida. The University of Miami. Division of Neonatology-Department of Pediatrics, presents its Annual Neonatal Symposium, "New Developments in Neonatal Respiratory Care," at the Doral Ocean Beach Resort. The symposium includes lectures, discussions, and a workshop on pulmonary function testing in neonates. Contact Charles R Bauer MD, Program Co-Director, University of Miami-Department of Pediatrics (R-131), PO Box 016960, Miami FL 33101, (305) 547-5808, fax (305) 547-3501.

March 3-5, 1993 in Lyon, France. The Journées Internationales de Ventilation à Domicile presents the International Conference on Home Mechanical Ventilation. The meeting includes scientific sessions, practical education sessions, and poster presentations and exhibits. Simultaneous translation (French/English) will be offered. Deadline for abstracts is December 1992. Write: JIVD (SRMAR) 93, Grande Rue de la Croix-Rousse, 69317 Lyon Cedex 04, France. Call (33) 78 39 08 43, fax (33) 78 29 98 94.

October 24-29, 1993, in Jerusalem, Israel. The XIVth World Congress of Asthmalogy convenes in Jerusalem. Topics to be presented include molecular biology in lung disease, cells involved in asthma, and house dust mite and asthma. Abstracts are currently being accepted. For more information, contact Gil-Kenes, 1617 JFK Blvd, Suite 946, Philadelphia PA 19103. (800) 223-3855.

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Banks, Ron E 1166
Barnes, Thomas A 1204
Beam, William R 1187
Bosso, Frank J 1197
Brougher, Pat 1209, 1211
Burns, Tom 1211
Carlson, Kathleen S 1193
Cimo, Darlene D 1206
Day, Sharon 1161
Douglas, Katherine A 1193
Ganetis, James A 1205
Hess, Dean 1209
Kigin, Colleen M 1209
Kollef, Marin H 1166
Krieger, Bruce P 1207
Lewis, Robert M 1153
MacIntyre, Neil R 1161
Maron, Michael B 1197
McCormack, Michael T 1166
Miller, Christopher C 1175
Miller, John WR 1175
Pierson, David J 1211
Shigeoka, John W 1205
Wanger, Jack 1187
Wooton, Larry 1161

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141	142	143	144	145	146	147	148	149	150	151	152	153	154	155
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